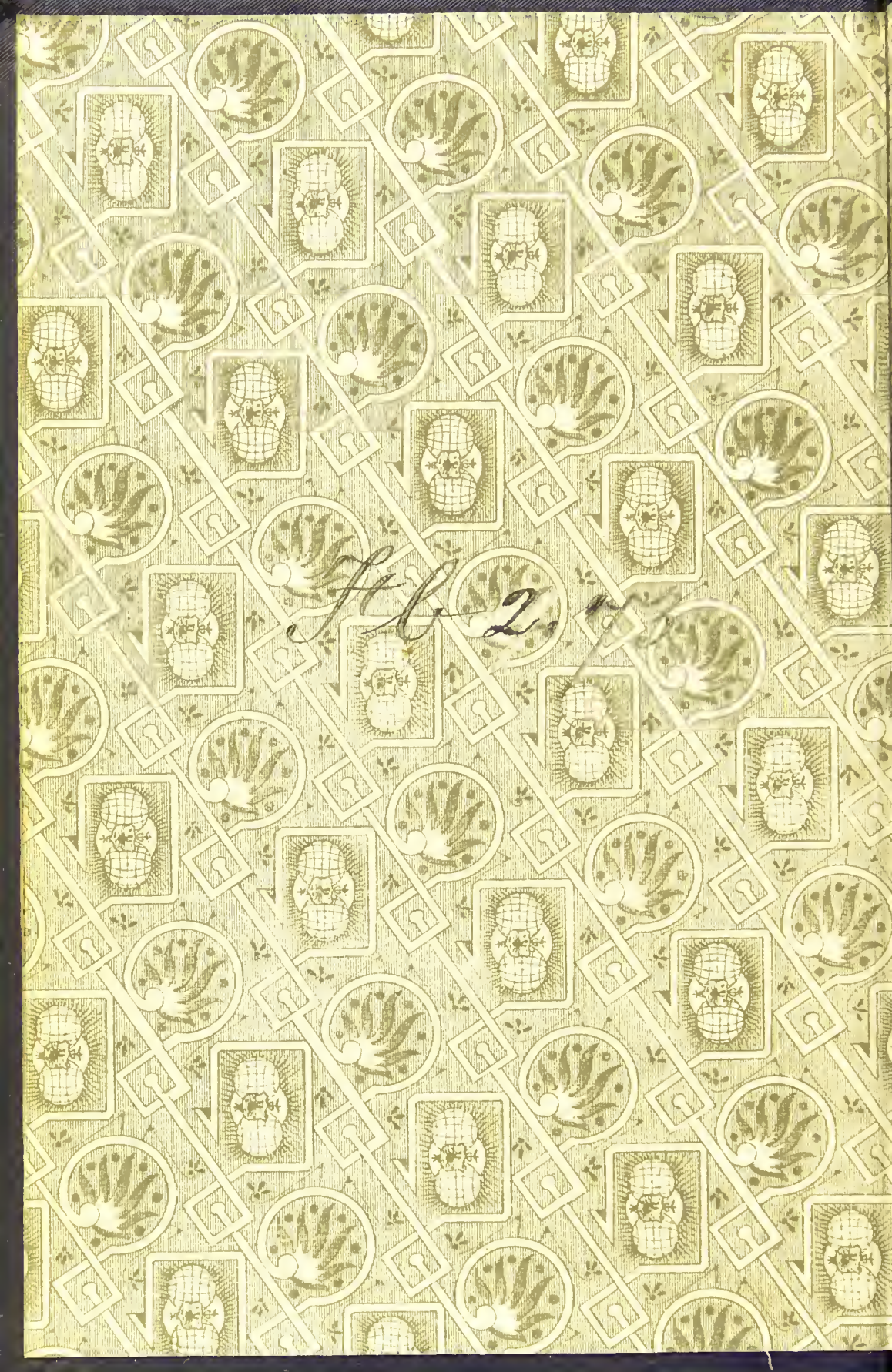

No. 7 IN THE PHYSICIANS' AND STUDENTS' READY REFERENCE SERIES.

EPILEPSY:

ITS PATHOLOGY AND TREATMENT.

BEING AN ESSAY TO WHICH WAS AWARDED A PRIZE OF FOUR THOUSAND FRANCS BY THE ACADÉMIE ROYALE DE MÉDÉCINE DE BELGIQUE, DECEMBER 31, 1889.

BY

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TO

DR. THOMAS B. BRADFORD,

SURGEON TO THE DELAWARE HOSPITAL
OF WILMINGTON,

I DESIRE TO DEDICATE THIS ESSAY,

AS AN EVIDENCE OF

AFFECTION AND FRIENDSHIP.

PREFACE.

This essay upon epilepsy was considered by the Royal Academy of Medicine in Belgium as worthy of a prize of four thousand francs, and this must be the chief excuse for its publication in the face of the large amount of literature daily accumulating concerning this important disease.

The author believes that it is fairly representative of the views held as most correct by the best minds of the profession, and has endeavored to separate the good material in literature from a vast mass of superstition and nonsense which persons even in our own generation have contributed. If this book can in any way help the physician in the present, or aid in the discovery of remedial measures capable of curing epilepsy in the future, its publication will not be useless nor a burden upon a profession notorious for its patience with authors.

222 S. 15th St., Philadelphia,
August, 1890.

EPILEPSY:

ITS PATHOLOGY AND TREATMENT.

EPILEPSY.

Synonyms.—*Latin:* Morbus sacer, Morbus vel sacer, Morbus major, Morbus herculeus, Morbus comitialis, Morbus convivialis, Morbus mensalis, Morbus insputatus, Morbus viridellus, Morbus vitriolatus, Morbus sontieus, Morbus carducus, Morbus unaticatus, Morbus foldus, Morbus sideratus, Morbus scelestus, Morbus demonicus, Morbus deificus, Morbus astralis, Morbus St. Valentis and St. Joannis, Analepsia, Apoplexia parva, Passio Caduca et Perditio. *French:* Epilepsie, Grand mal, Haut mal, Epilepsia. *German:* Fallsucht, Epilepsie. *English:* Epilepsy, Falling sickness, Fainting sickness, Fits. *Italian:* Epilepsia. *Scandinavian:* Epilepsin fallendsot.

Definition.—Epilepsy is to be defined as a disorder of the nervous system characterized by sudden convulsive seizures of temporary duration, the muscles of the parts affected being first in tonic spasm, then alternately contracted and relaxed (clonic spasm), the attacks generally occurring at irregular intervals, and being always accompanied by loss of consciousness, more or less complete, in the typical disease. The movements also have no relation with those of ordinary life. In rare instances, however, one or more of these symptoms may be absent, and yet the disease be epilepsy.

Before the writer goes further he must endeavor to make clear the meaning of the terms generally employed in discussing this disease, in order that in using a given term his meaning may be well defined.

It is an evidence of the paucity of our knowledge in these convulsive disorders that the nomenclature employed is at once inaccurate and inexpressive even of the little we know. Thus, many writers divide the disease into idiopathic epilepsy and organic epilepsy, because in the one case we have not been able with the means at our disposal to discover any changes from the normal in the parts when they are examined, and in the other we find more or less gross lesions. It is to be hoped that before long this ignorance may be enlightened, for it is hardly possible to imagine that a given number of cells can evince morbid tendencies for years and still remain organically normal. Some changes must occur which we are not quick enough to discover. The term *organic epilepsy* is used as well to designate not only direct morbid change in a given set of cells, but to identify indirect perversion of their function produced by the irritation or pressure or interference of nutrition by a neighboring and demonstrable neoplasm. Of this more will be said under the heading of Pathology.

History.—Almost as far back as we have records of events the story of the disease called epilepsy reaches. Long before medicine, as we know it at the present day, took the shape which separated it from witchcraft and sorcery, civil writings incidentally spoke of it, either describing the disease in detail, or giving it a name which in its meaning described the affection. Long before the time of Galen and Hippocrates we find mention of its character, and the famous Greek just mentioned has described it with a characteristic accuracy which

seems as true to the disease to-day as it was hundreds of years ago.

Almost every century since their time has borne in its medical annals some account of its symptoms, and probably no disease has ever given rise to more discussion, both medical and otherwise, than the one before us; for, unlike those diseases of an epidemic character which from time to time swept off thousands of the human race, epilepsy, by its constancy at all times and in all places, fastened itself upon single individuals and left accurate impressions on the minds of its observers, who were not disturbed by the fear of contagion and who therefore wrote nothing hastily. The very fact that epilepsy asserted itself in the bodies of its victims at the most inopportune times and before all men caused it, too, to be brought to the notice of the people more than other affections even more wide-spread, but which by reason of their hidden nature were less frequently seen. It is illustrative of the true birth of medicine that epilepsy is rapidly passing from the cloud of ignorance into the light of modern science. For over twenty centuries it has passed among men as a something too intangible to explain, too far beyond their power of treatment to yield to any one, however skillful he might be; yet in the past twenty years, at the most, more progress has taken place in our knowledge as to its entire course than in all the preceding centuries. If we glance back among the older writers we find that they tried new remedies as frequently as they failed in treating the disease, and one after another advanced hypothetical conclusions as to its causation in almost every case, many of which were foolish even in the light of their own knowledge. Every form of explanation was attempted; the clergy, the laity, and the most

ignorant of the common people shared the universal privilege of inventing new theories and therapeutic measures, and yet not one opinion has survived, and our building of knowledge of epilepsy contains no stone save those gathered in the last century.

One of the first efforts made by the earliest workers of the present time was the denuding of epilepsy proper of that enormous amount of surrounding drapery partaking of an epileptiform character, but at present recognized as arising from entirely different causes. Spasm, like dropsy, has come to be considered as a mere evidence of some condition more or less defined, and not as a disease itself. It is the result, not the cause. The chief reason for all this improvement lies in the increased desire for knowledge, which has brought about a vast amount of original research in this field as well as in others, and it has been well said by the famous German physician, Nothnagel, that no affection of the human race could be held up for which so much has been derived of value from vivisection and experiments on the lower animals than the disease now before us.

So much is to be said of the disease as we at present know it that the writer cannot linger over the writings of those of long ago, which, while interesting, are but gropings in the dark and require much space, and we shall pass over to the last few centuries, during which faint glimmerings of light have begun to creep in. Early in the sixteenth century the first reports of methods of cure of a rational character appeared, and for many years formed almost entirely the greater part of the literature of epilepsy. Hector Boethius [1] in 1536 wrote of castration as a method of cure, not only directly but indirectly, to prevent hereditary tendencies. According to this

[1] Cronikles Scotland, Edinburgh, 1536, lib. i.

writer, the custom of castration with this object in view existed for many years among the Scotch previous to the time at which he wrote. Thus he states that, while this treatment was accorded to epileptic males, epileptic females were isolated, and if by chance they conceived, both the mother and the child were to be killed. Jean Taxil [1] also noted this means of cure as long ago as 1603, but Platenus, Mercatus, and Heurnius,[2] along with Coelius Aurelianus,[3] performed the operation many years prior to these writers, and for the same purpose, namely, of curing the disease, which, in one of its forms, was generally believed in those days to be dependent on the retention of semen, which, undergoing corruption, produced reflex convulsions by the irritation set up.

Very early in the history of medicine, however, these views were largely held, for Arethaeus [4] asserts that many physicians, among them the celebrated followers of Æsclepius, thought that venery cures the epilepsy which begins at puberty. The same opinion was held by Scribonius Largus, and the retention of semen was the exciting cause in the belief of all. It would seem, however, that physicians of that day were as quick to contradict one another as they are now, for Alfarius á Cruce contended that the epilepsy of puberty was an incident of the age of the individual, not of the retained semen, denying that sexual intercourse ever gave relief except in very rare instances. Unfortunately, this writer does not seem to have impressed this teaching on his pupils, for one of them, Sinabaldi,[5] declared coitus to be a useful means

[1] Traité de l'Epilepsie, etc., Tournon, 1603, p. 229.

[2] Opera Omnia, postrema editio, Lugduni, 1658; de Epilepsia, chap. xxiii, p. 421.

[3] Morb. Chron., lib. i, cap. iv.

[4] Opera Omnia. Morb. Chron., lib. i, cap. iv.

[5] Genecanthropia, Romae, 1643, p. 886. C.

of eure in some forms of epilepsy, but not in all forms. Tissot,[1] who wrote, eomparatively speaking, mueh more reeently, argued that this retained semen, by eorruption, produeed epileptie attaeks, and by so doing brought one of the oldest superstitions down to modern times. In justiee to the older writers, however, it should be said that they did not carry out these ideas to such an extreme as to lose sight of the faet that other causes might produce the disease. Indeed, Galen and others reeognized that sexual exeesses might have similar results with sexual eontinence. It should be remembered, too, that epilepsy and eoitus were supposed to be elosely assoeiated, for epilepsy often eame on during intereourse; so that it was eompared by Demoeritus to a "seizure":—

"μικρα επιληχια;"

or, as Faustus has deseribed it:—

"Turpis, et est morbi species horrenda eadueo."

The following extraets in regard to the more modern superstitions will also be of interest to the reader, as showing how barbarous curative measures were employed in the eighteenth eentury[2]:—

To Monsieur the Criminal Lieutenant-General and President of the Seneschal's Court of Lyons:—

Sieur Claude Pessoneaux, mereer of that town, humbly prays and assures you that, for eight years, he has been subjeet to epilepsy, whieh attaeks him almost daily, even several times a day, and entirely deprives him of eonseiousness. And as he has heen informed that several medical authors promise a eure from the administration of a speeifie remedy, eomposed of various roots and the skull of a man who has died a violent death—a remedy whieh, when properly prepared, has the reputation of being most effieaeious, aeeording to the eertifieates whieh M. Hedoin, physieian to the king, and *agrégé* of the College of

[1] Traité de l'Epilepsie, Lausanne, 1785, p. 73.
[2] Rev. Méd. et Chirurg. de Paris, November, 1855.

Medicine at Lyons, and the Sieur Aubernon, surgeon of Lyons, have to-day given under their seal, which certificates your petitioner shows and sets forth to you; and as he has been informed that a criminal, condemned by your judgment to be hanged, is about to be executed, may it please you, Monsieur, to permit him, after the execution of this miserable person, to remove the skull of his head, in order to employ it in the preparation of this remedy; and he will consider himself bound to continue in prayer to God for your prosperity and health.

PESSONEAUX.

Let this be shown to the Procureur-Général of Lyons.
30th Octobre, 1706. CLARET LA TOURRETTE.

On behalf of the king I do not forbid the concessions of the present request [Signature illegible.]
LYONS, 30 Novembre, 1706.

Let it be done according to the decree of the Procureur-Général.
LYONS, 30 Novembre, 1706. CLARET LA TOURRETTE.

To Monsieur the Criminal Lieutenant-Général in the Seneschal's Court of Lyons:—

The sisters, rector, and governors of the General Almshouse in Lyons humbly pray and assure you that they require in pharmacy several skulls of persons who have died violent deaths, in order to use them in the composition of several very necessary remedies,—among others, of one against epilepsy, to which several persons in the above-named house are subject.

They are, therefore, obliged to have recourse to you. May it please you, Monsieur, considering the necessity above pointed out, and for the good of the poor, to permit the surgeon of the aforesaid house to select and remove from the burial-place of the Penitent Sisters of Mercy such skulls as they may find proper for the preparation of the aforesaid remedies; and to this end the said Penitent Sisters be invited to permit the said memorial, and the poor of the said Almshouse will pray for your prosperity and health.

BOURG, CHRISTIN.

Let it be done as it is required.
LYONS, 13 Septembre, 1710. CLARET LA TOURRETTE.

Another old theory resembled very closely that by which insane persons received the designation of "lunatic," namely, that epilepsy was always worse during the moon's phases. This notion was held until very recent times, when M. Moreau proved it to be untrue by an

enormous collection of evidence against it. Thus, he saw no less than 47,637 fits, of which 26,313 were between the phases of the moon and 16,324 during its phases.

Even at the present day superstitions are very rife among the lower classes, and all kinds of filthy decoctions are employed against epilepsy. The writer is told that in Melbourne, Australia, snakes' heads in rum have held until very lately a high reputation for the relief of epilepsy. Even supposedly sane individuals of rank at the present day are guilty of the most wretched superstitions. The following is of interest, as showing this, taken from an address by the President of the Eckenfoerder Shooting Club to its members:—

> Her Highness, Princess Bismarck, wishes to receive, before the 18th inst., as many magpies as possible, from the burned remains of which to make an anti-epileptic powder. I permit myself, high and well-born sir, to entreat you to shoot as many magpies as you can in your preserves, and to forward the same to the Chief Forester, Lange, at Friedrieksruhe, or hither.

Symptoms.—One of the first and most marked symptoms of an oncoming attack of epilepsy is a peculiar sensation felt in some portion of the body, generally below the brain, which gradually rises up over the patient, either rapidly or slowly, like an oncoming cloud, until, the head having been reached, the patient is immediately convulsed and unconscious, and almost instantly is seen to be in the very acme of the nervous storm. Simultaneously with the arrival of the aura in the cervical region the person utters a peculiar cry or scream, so wild, so harsh, and so characteristic that it has been called the "epileptic cry," being probably due not so much to a voluntary impulse as to a sudden expulsion of the air from the thorax by the convulsive contraction

of the abdominal muscles, as well as those of the thorax, and its rapid passage through the glottis narrowed by rigid spasm of the muscles governing this opening. Synchronously with this cry the muscles of the whole body, in a widespread attack, become strongly contracted until they are in a tonic spasm, and then, having momentarily relaxed, pass into alternating relaxations and contractions, which throw the sufferer now to this side, now to that.

With the tonic spasm the muscles of the face often produce hideous distortions of the features, in some cases bringing about the so-called *risus sardonicus;* the head may be drawn to one side, and under these circumstances the eyes are generally turned in the same direction; the jaws are locked one against the other, and the lower jaw may also be drawn away from the median line of the face in the same direction as the eyeballs. Sometimes the whole body is rotated. In the 970 cases[1] analyzed by the writer complete rotation to the right is mentioned as being present in 49 persons, and to the left in 52 cases. There is, therefore, no difference worthy of note in these numbers.

The arms are strongly flexed at the elbows, while the hand is still more strongly flexed at the wrist; the fingers are also so bent into the palm of the hand that not unfrequently the skin in this region is found indented by the nails. To speak briefly, the arms, legs, and body are drawn and jerked in the direction of the most powerful muscles, and, as a consequence of this, opisthotonos, during the tonic stage, is by no means uncommon. Exceptions to this rule do, however, frequently occur, and when present show that the paroxysm is exerting its chief influence on the weaker muscles, while the stronger

[1] University Medical Magazine, 1889.

ones are affected at least to a less degree. As a general rule, too, the muscles of one side suffer more than those of the other. Unfortunately the writer finds, in the cases collected by him, that in only 158 instances were any remarks on this point made. In these 158 the right side was most affected in 77 cases, and the left side in 81 cases. It is evident, therefore, that both sides suffer about equally. The author has not attempted to analyze these cases as to relative frequency of the general symptoms, as it would hardly be justifiable, for in some of the cases they may have occurred and the reporter neglected to name any one of them.

The legs may be firmly flexed on the abdomen, while the fingers are rigidly extended. The change in the color of the face is very marked and almost typical of the disease, being at first pale, then flushed, the flushing deepening often into a livid purple, owing to the asphyxia produced by the convulsive contraction of the thorax. In some cases the eyelids are widely drawn apart so that the eye, owing to its fixation, has a staring appearance; in others they are so tightly closed that the fingers of the on-looker can scarcely force the lids apart. The staring but blank expression of the eyes is also increased by the slow dilatation of the pupils, which always accompanies the asphyxia.

The duration of these tonic contractions rarely exceeds two minutes, and in most cases is limited to but a few seconds. It is followed by clonic spasms, already described, which are ushered in by more or less violent tossings, but whose onset is forewarned by peculiar vibratory thrills, which run through all the affected muscles. The eyelids tremble, the body changes its position ever so slightly, and then, as if the vibrations gained greater and greater power with each moment the fibrillary trem-

ors give way to muscular contractions. The expression of the face, which in the preceding stage was set and firm, is now constantly changed by the movements of the facial muscles; the jaws, no longer locked together, are gnashed and crunched one upon the other; the tongue is alternately protruded and drawn back, and, as a consequence, is often caught between the teeth and bitten and lacerated. The excessive movements of the muscles of mastication force the increased quantities of liquid secreted by the salivary glands from the mouth in the form of froth, which is often stained with blood by reason of the injuries to the tongue. The constancy of the convulsive movements now becomes less and less marked; well-developed remissions occur between each toss of the body, until the movements cease entirely; but it should be constantly borne in mind that the prolongation of the remissions does NOT produce *any decrease* in the severity of the intervening spasm, the final spasm often being even more violent than the first.

The intense discoloration of the face begins to pass away as soon as the remissions, by their length, permit the blood to be oxygenated, its disappearance being temporarily arrested by each paroxysm. Finally, the spasms having ceased, the patient lies before us relaxed, unconscious, and exhausted, and passes into a deep sleep or coma, which lasts a variable length of time, and from which he cannot be aroused except very rarely, and then with great difficulty.

Absolute coma was recorded in the writer's collection of cases as present in 104 cases, in 12 others it was marked as absent, and in the remaining cases the person keeping the record failed to report concerning it.

Even when the sleep has passed away the brain is evidently disturbed in its functions for some hours or

perhaps days, and headache is, not rarely, complained of after the patient seems like himself in other respects. The sphincters are very rarely relaxed, although the urine may be voided, as may also the fæces. According to Gowers[1] this relaxation is more commonly associated with nocturnal epilepsy, and this opinion coincides with the writer's experience.

That urinary incontinence is extremely rare is shown by the fact that in the cases collected by the writer (970) it was only recorded as occurring in 45 cases.

Having described a typical attack of epilepsy, let us proceed to an analysis of the various symptoms detailed. Very commonly in epilepsy we find that the patient is covered, during or after the paroxysm, by a profuse sweat, which has been assigned by some to the excessive muscular movements and by others to vasomotor changes which they think occur. Emminghaus[2] has made reports and studied this matter in order to determine which of these theories is the true one, and inclines to the belief that it is due solely to vasomotor disturbance, but it is probable that both the muscular movement and the changes in the circulatory system are at fault. In some cases the muscular movements may amount to almost nothing, and under these circumstances any sweat must be due entirely to a disordered peripheral circulation and its nerve-supply, or to disturbances in the nerves governing the sweat-glands. Those who believe completely in the neurotic origin of the sweat point out very forcibly that often in the most severe fits no sweating takes place.

The Aura.—One of the most interesting and important of all the symptoms described is the so-called *aura*, and some difference of opinion has arisen as to the fre-

[1] Diseases of the Nervous System, vol. ii, p. 681.
Arch. f. Psychiatrie, 1874.

quency of its occurrence, some authors stating it to be very rare, while others see it very constantly. There can be little doubt that in many cases it is as constantly present as in others it is absent, and it would appear that the nationality of the subject has something to do with the occurrence of this signal of the attack; at least, if we may judge by the statements of the chief authors of each nation. Thus, in America Wood[1] states that "the aura is wanting in a very large proportion of the cases of true epilepsy," and Hammond agrees with him.[2] In England Gowers states it to occur in about one-half the cases, and Bristowe[3] states it to be not uncommon. In France and Belgium the aura appears to be present in more than half the cases, in one form or another, as it is also in Germany, according to the most prominent neurologists.[4] In the 970 cases collected by the writer it was found that the aura was recorded as present in 362 cases and absent in 138 cases. In the remaining 470 cases the occurrence of an aura was not mentioned.

The following table shows the character of the aura in the cases where it was recorded :—

Tingling, 45.	Dizziness, 15.	Chilliness, 7.
Visual, 27.	Numbness, 15.	Laughter, 4.
Pain, 26.	Disturbed respirations, 14.	Drowsiness, 3.
Twitching, 21.	Gastric, 15.	Faintness, 3.
Epigastric, 20.	Cramp in muscles, 11.	Abdominal, 3.
Headache, 16.	Olfactory, 11.	Aphasia, 3.
Auditory, 16.	Mental, 8.	Unclassified, 44.

The word *aura* is derived from the Latin, signifying *vapor*, and its application to certain symptoms of epilepsy arises from the old Greek theory that the fit began

[1] Nervous Diseases and their Diagnosis, p. 103.
[2] *Ibid.*, p. 682.
[3] Theory and Practice of Medicine, 6th ed., p. 1114.
[4] Nothnagel.

by the ascent of a vapor in the veins of the extremities. In later times it was imagined that the nervous impulse causing the spasm arose in the part where the aura first appeared, since the attack could be put aside by the tightening of a ligature around the arm or leg; but this is held by most of the students of the disease at the present day to be impossible, since the application of a ligature, where the convulsion is due to brain-tumor, may stop the onset of the paroxysm. It cannot be gainsaid that this is true, but while the lesion may be present in the brain it is no reason that the impulse for the convulsion may not arise peripherally by a species of reference of the irritation to that part, and in the so-called reflex epilepsies there can be no doubt that the impulse is peripheral, not centric. In the writer's own practice he has seen a case which to a certain extent contradicted the opinion that the impulse does not ever arise except in the centre. Thus, in a case of adherent prepuce the aura always began in the penis, and the attack could be put aside by tightly grasping that organ. Circumcision cured the epilepsy, which could not therefore have had its origin centrically; further than this, the whole list of reflex epilepsies show that the aura and the cause of the attack may exist in the same part of an extremity. Then, too, we have nothing as yet to prove that it is impossible for a centric nervous lesion to produce such functional changes peripherally as to cause disease in that part. Every one knows how disease of the brain may cause descending degeneration in the spinal cord, or ascending disease of the cord produce brain-lesions, and direct physiological experiment has proved that a nervous lesion in the periphery may cause not only centric changes but peripheral changes elsewhere entirely separate from the part primarily injured; as, for ex-

ample, the famous experiments of Brown-Séquard, where section of the sciatic nerve in the leg of a guinea-pig produced structural alterations of the skin of the face, and in which an epileptic attack could be brought on at any time by pinching that area, or the disease cured by removal of that portion of the skin.

It will be remembered that, as a general rule, receptive nerve-centres refer impulses to their peripheral nerves; as, for example, the pain in the knee and ankle in coxalgia, or the stomach-pain of vertebral disease; and so may the irritation of a brain-tumor be referred to the periphery, and, gradually increasing, cause a convulsion by sending to the motor centres irritating messages. This theory is also supported by the fact that the area involved in the brain is accurately pointed out by the point of origin of the aura. If, for example, the aura is in the hand, the hand-centre is probably diseased.

The writer has stepped aside to discuss this point a little more fully than is, perhaps, necessary, were it not that, while he is a firm believer in the idea that all epilepsies are essentially centric, he fears that there is a tendency just at present to regard with suspicion the occurrence of reflex epilepsy arising peripherally.

The aura, or warning, while possessing general characteristics in common in all cases, is by no means identical in each individual. By far the largest number of cases, where it is present, have it in an extremity, and, if it be not there, then it is probably in the stomach; while it is not uncommon to see persons suffering from epilepsy who have as an aura a general, indefinable sensation all over the body. In much more rare instances the auræ are situated in the organs of special sense, and are evidenced by sudden attacks of blindness or of deaf-

ness. It is worthy of note, however, that whereas the auræ may differ in every case in origin, seat, and limitation, they are remarkably constant in the same individual, rarely, if ever, changing unless to grow more or less well defined. A careful analysis of an enormous number of cases by hundreds of observers shows that the aura most commonly met with is that beginning in the hand; next, that beginning in the leg or foot; next most common, that arising in some of the viscera, and after these those which arise in the face and tongue. The rarest form of aura is that which arises in the sides of the trunk.

Not only may the seat of the aura be various, but its sensations may be even more aberrant. Undoubtedly the most common sensation is the indescribable sensation of a vapor or cloud, already spoken of, but in a large number of cases the sensations are described as being quite painful, or perhaps as partaking of the feeling that the part is in active movement when in reality it is still quiet. Others speak of it as a sensation of cold, others of heat or burning, and still others of trembling and indescribable distress. In certain cases the sensation is confined to the spot where it is first noticed, and fails to travel upward or toward the central nervous system. When the seat of the aura is in the thorax or abdomen, it frequently produces, as it travels upward, a sense of strangulation, which is only a seeming arrest of respiration, since in the slowly-moving auræ the glottis is not closed until some moments later. Auræ in these regions are nearly always associated with the distribution of the pneumogastric nerves and the respiratory portions of the spinal accessory nerves. Generally the visceral sensations occur in the middle line, rarely to the left of that line, and

scarcely ever to the right. A very curious fact in regard to the abdominal auræ is, that when pain is felt in the epigastrium it never ascends to the head, but remains in this region till the convulsion comes on, whereas if the sensation is not one of pain it frequently extends to the cerebrum. Ch. Bonet[1] mentions an interesting case of a man of 50 years, who had an epileptic aura consisting in a swelling of the groin.

The vagus nerve, as has already been stated, seems to carry out a large part of the sensation of the aura, both in its gastric branches and respiratory filaments. The cardiac filaments also show signs of being concerned by palpitations, pain, or cardiac discomfort.

In the aura associated with the nerves of special sense the most common perversion of function has been found to be that of sight, which, according to Charcot and Gowers,[2] is twice as frequent as all the other special-sense auræ put together. It may consist of a single or, more commonly, many colors, floating before the eyes, red and blue being those most usually seen (indeed, no other color is ever seen alone), or it may consist in a sudden diminution or increase in the size of all surrounding objects. In others the ocular symptoms exist, but are entirely beyond the ability of the patient to describe, while diplopia, blindness, or complex visions may be present. Thus, in some cases, the vision of an old woman or man, holding in the hand a hammer, with which a blow is about to be struck, has ushered in an attack and all succeeding attacks. One of the oldest reports of such cases that we have is one of Joannis Schenckius,[3] who, in 1665, recorded the instance of

[1] Sepulcretum Anatom., lib. i, sect. xii, p. 291.

[2] *Loc. cit.*, p. 684.

[3] Observationum Medicarum Rariorum, Frankfurti, 1665, lib. i; de Epilepsia, p. 104.

a young man who always saw a woman offering herself to him in a lascivious manner before each fit, which was accompanied each time by an emission of semen. In other cases flashes of light and sparks may pass before the eyes, or objects seem to move toward the patient more and more rapidly, and as they reach him he is thrown down by the convulsion. Contrariwise, the objects may appear to be leaving him, and as they grow dim in the distance the paroxysm seizes the patient. The writer might go on enumerating the various ocular disturbances almost indefinitely were it not that it would be useless and tiresome to the reader, who, after what has been said, must have been impressed with the idea that any vision or aberrant movement of the apparatus vision may occur.

Auditory disturbances are also frequently present as auræ. Thus, there may be a whizzing or buzzing sound, or a crashing, which grows louder and louder until the patient falls; or, instead of these, a desperate stillness which the loudest sound cannot dispel, and which cannot be distinguished from ordinary deafness save in the transient character of the loss of hearing. Short peals of music sometimes float the patient into an attack. Drums beat martial airs and fifes may seem to play.

The third form of perversion of special sense is that of smell. Thus, in one patient there may be an odor of phosphorus, in another of verdigris, in a third of some common or rare drug. Sometimes the subjective odors are agreeable, sometimes disagreeable, although, even if the odors are in themselves not unpleasant, they speedily become so, owing to their association in the patient's mind with the disease.

The infrequent forms of special-sense auræ are those

of taste, and in these the gustatory apparatus may perceive flavors pleasant or vile, sometimes bitter or metallic.

Still one other seat may give rise to an aura, namely, the brain. Psychical auræ, as they are called, almost always consist in an intense feeling of alarm and terror, or they may consist, in very rare cases, of a certain intellectual thought or idea. In this form of aura the idea of an imminent danger to the person by reason of an apparent threatening act of a bystander may drive the patient to inflict a blow in self-defense which may be fatal, but of which, after the fit, he has no recollection. Of this I shall speak when considering epilepsy in its relation to crime. So dreamy is the mental condition in some cases that, while the patient is conscious of the passage of ideas, he cannot, either before or after, express what they were. Hughlings-Jackson calls this the "voluminous state." Probably the most uncommon symptom is that given by Gowers, and consists in a sensation that something is morally wrong.

The cephalic auræ vary quite as much as those found elsewhere, and may be evidenced by giddiness, vertigo, fullness of the head, or sensations of heat or cold either within or without the skull. Nausea may attend the vertigo if it is prolonged.

The frequency of this symptom may be judged from the following table, and the relative frequency as compared to the other symptoms is also to be found on pages 22 and 23 :—

Vertigo.	Males.		Females.		Total.		Per Cent.	
Doubtful, . . .	20	25	22	23	42	48	60	68
Absent, . . .	5		1		6		8	
Present occasionally,	10	11	6	10	16	21	23	30
Present frequently, .	1		4		5		7	
	36		33		69		98	

When we exclude the doubtful cases, we find that vertigo is present in 77 per cent., and that more females are affected than males, in the proportion of 90 to 68.[1] In the cases collected by the writer vertigo is recorded in 206 cases as present, and in 18 cases as absent. In the remaining cases no mention of vertigo is to be found.

Having now considered to a very wide extent the chief characteristics of the auræ of sensation, let us turn to those manifesting themselves by motor disturbances. In some such instances contractions of the muscles of one or more fingers gives the warning, or, indeed, the entire limb may be moved, although this is much more rare. In the same manner the attack may give notice of its approach by spasm of the face, particularly in the zygomatic muscles, and sometimes in the *orbicularis palpebrarum.*

A very close relationship exists between the onset of the attack itself and the aura in these cases; that is to say, it is difficult to decide how far the contraction of a finger partakes of the character of an aura and how far it partakes of the primary movements of the convulsion, for the spasmodic movements, in some cases, pass from muscle to muscle until the entire body is convulsed. It is also as true of these motor auræ as of the sensory variety, that they indicate the place in which lies the centric trouble, if centric trouble exist, and it is equally undeniable that these motor auræ generally accompany, not the ordinary idiopathic epilepsy, so called, but that dependent upon a localized injury, or a tumor of the brain. On the other hand, it is equally certain that they do partake of the character of an aura, since forcible extension of the fingers, flexed by

[1] Reynolds on Epilepsy. London.

a motor aura, will in many instances avert the attack in the same manner as will a tight ligature around the wrist in the case of a sensory warning. Still other cases exist than those mentioned, in which a combined aura may be present, commonly made up of a motor and sensory aura, or of a motor and special sensory aura, although all these may occur in a single case.

Precursory Symptoms other than Auræ.—When speaking of psychical auræ the writer should, perhaps, have spoken of the apathy of the mental processes which sometimes precedes an attack, were it not that this symptom and others like it are more like the prodromata of a disease than a sudden and brief warning. On the other hand, in place of apathy there is sometimes for several days before a fit a great increase in the rapidity of thought, ideas chasing through the brain so rapidly that they are lost before they can be put into words. In certain cases this mental condition finds itself expressed in bodily restlessness, so that the man may be driven to take long walks, or roam about the streets. Violence may be indulged for several days to such an extent as in no way to separate itself from that of mania except that there is generally a sane condition in regard to other matters. While in this state, persons who are innocent and total strangers may be attacked by the patient, or furniture demolished by him. Children occasionally for a few days before a paroxysm become ungovernable, often attempting to bite their playmates and attendants. Great fear of an impending evil sometimes is present, or is supplanted by the most abject melancholia. Still other cases are recorded in which an extraordinary loquacity asserted itself in men of a commonly morose or taciturn nature, while sometimes such imperfections in speech occur for a few days

as almost to amount to aphasia. The touch may be hyperæsthetic, or diplopia or hemiopia exist for twenty-four or forty-eight hours beforehand. Bleeding at the nose or profuse salivation, with tenderness of the gums, may also be forms of prodromata, and several cases are on record where the man has suddenly blown his nose at dinner-table, and then, springing up, dashed into the street. Other men have been seized with a violent desire to strip themselves of clothes, even in the street or at a theatre.

Quite a number of statistics have been collected by various clinicians as to the relative frequency of the precursory symptoms of epileptic attacks, chief among which should be mentioned those of Delasiauve,[1] which are here given. In 264 cases, where precursory symptoms were always present, he finds reason to divide them into seven groups as follows :—

Seventy-five Cases with Precursory Symptoms in Head.

Vertigo, flashes of light,	23
Headache or feeling of weight in head,	15
Heat of face,	3
Various localized sensations,	13
Indefinite,	1
Illusions, hallucinations,	9
Rotation of head and eyes,	5
Grinding of teeth, with loss of movement of tongue,	2
Somnolence,	1
Constriction of throat,	3

Twenty-two Cases with Precursory Symptoms in Thorax.

Oppression and sense of suffocation,	9
Feeling as of a ball in throat and chest,	2
Shivering with cold,	5
Pain or heat,	4
Palpitations, spasms,	2

[1] Traité de l'Epilepsie, 1854, p. 47.

Thirty-two Cases with Precursory Symptoms in Abdomen.

Pain, eructations, and vomiting,	13
Intestinal or uterine colic,	3
Sensation of a ball,	3
Sensation of cold or vapor,	6
Stomachal heat,	1
Undefinable sensations,	6

Ninety-four Cases with Precursory Symptoms in the Extremities.

Numbness, contractions, jerkings, retractions, cramps, and formications,	36
Pain, with or without spasm,	13
Tremblings,	10
Aura or similar phenomena,	20
Undefinable sensations,	15

Twenty-two Cases with Precursory Symptoms of an Undefinable Character.

General agitation and rotation of body,	8
Condition of discomfort, fainting,	6
Vague sensations,	7
Moroseness,	1

Five Cases with Precursory Symptoms Situated in the Genital Organs.

Retractions of testicles or aura starting there, sensations in the uterus,	5

The seventh series contained those rare cases where there was a desire to defecate, urinate, or in which profuse sweating came on.

In a collection of cases made by Hammond,[1] in 128 epileptics out of 286 persons suffering from the disease, there were precursory symptoms.

When we summarize Delasiauve's results we find that the largest number of the precursory symptoms

[1] Diseases of the Nervous System.

occurred in the extremities (94 cases), the next largest number in the head (75 cases), next in the abdomen (32 cases), and last of all in the thorax (22 cases), the remaining 27 cases in the sixth and seventh somewhat artificial divisions of the author quoted being of an undefinable character.

Addison has also studied the relative frequency of the symptoms of the attack itself, with somewhat interesting but in no way surprising results[1] :—

	Males.	Females.	Total.
Loss of consciousness,	29	10	39
Muscular cramp,	10	1	11
Bit tongue in fit,	21	6	27
Uttered cry before fit,	25	5	30
Fell during fit,	39	9	48
Wet bed during fit,	19	7	26
Wet bed at night only,	12	3	15
Wet bed night and day,	27	8	35
Rotated in fit,	4	1	5
Convulsions on right side,	10	3	13
Convulsions on left side,	8	2	10
No coma after attack,	2	1	3

Of course, many of these symptoms occur in a single individual in many instances, but the table serves to illustrate what has been said in regard to the frequency of certain signs, such as the loss of consciousness, the falling to the ground, and the frequency of coma after a seizure.

The Pupil.—The dilatation of the pupil, which comes on in nearly all epileptic attacks, has connected with it the interesting question as to whether the mydriasis is produced by the convulsion directly, or indirectly by the resulting asphyxia, or by both causes. The latter is probably the true answer, because it is well known to physiologists that the pupil generally dilates in con-

[1] Addison, Journal Mental Science, vol. xii, 1867.

vulsions, and also that asphyxia produces similar results by its action on the centric nervous ocular apparatus. In some cases it would seem that the nerve-storm continued in the ocular centres after all else is quiet because of the alternate contraction and dilatation of the pupil; but this will be referred to further on.

The writer has already spoken of the involuntary voidance of urine and fæces by epileptics, and when doing so stated that it was very unusual. It is not dependent, when it does occur, on the loss of consciousness, for it never occurs in some patients whose intellectual faculties are entirely lost, and does take place in others whose minds are merely dimmed for a moment. In these the spasmodic movements probably affect the walls of the bladder or its sphincter, or the walls of and sphincter of the lower bowel.

The Circulation.—Curiously enough, the circulatory apparatus escapes almost entirely in epilepsy; the best authorities, indeed, all agree in stating that the only changes are those brought about by the accompanying asphyxia. Voisin has published a curve showing that no change occurs during an aura, but Moxon, on the other hand, has asserted that a stoppage of pulse occurs in some instances, and, although many have denied this, one can readily believe that it may occur when the inhibitory cardiac filaments of the vagus are affected. Every one agrees, of course, that during the violence of the muscular movements the force and rapidity of the circulation is increased, and particularly the arterial pressure. The latest and most elaborate studies on the epileptic pulse, with which the writer is acquainted, are those of Mons. V. Magnon [1] who has shown that during the clonic stage of the convulsion the arterial pressure

[1] l'Epilepsie Paralytique, 1881.

is increased to a very great extent, as well as the pulse-rate, but that during the first or tonic stage the pulse-rate falls, and the rhythm is so altered that a complete systole and diastole may occupy six times the normal period. Afterward the pulse passes to the normal or into a condition of increased force and frequency.

Status Epilepticus.—Before passing on the writer should speak of the condition known as the "status epilepticus," in which convulsion follows convulsion so rapidly that consciousness is never regained, but the patient dies within a few hours as a result of exhaustion or asphyxia.

Probably the most thorough study that we have of this condition is that of Bourneville,[1] and his results are certainly worthy of introduction here. He details the symptoms as follows: État de mal epileptique is characterized by—

1. Frequent repetitions of attacks so close together as to be almost if not continuous.

2. By variable degrees of collapse, which may deepen into coma.

3. By more or less complete hemiplegia.

4. By increased frequency of pulse and respiration.

5. By elevation of temperature persisting even between the attacks.

As the case goes on the convulsions are replaced entirely by coma, or, in rare cases, violent attacks of mania may develop. In this state the body rapidly emaciates, bed-sores develop, and death ensues from exhaustion.

It is noteworthy, however, that with the development of the bed-sores the temperature rises again as high as during the convulsions.

[1] Bourneville, État de mal Epileptique, 1873.

Time of Day.—In an analysis by Boyd,[1] to determine whether the seizures of epilepsy were more frequently at night or during the day, he found that in a collection of 3202 fits 1962 occurred in the day-time and 1240 at night, showing that the waking hours are most commonly interrupted.

Irregular Symptoms.—So far only the more regular symptoms of an attack have been given, and the writer would not be doing justice to the reader to let him think that all cases of epilepsy are so fully accompanied by a long train of constant signs as have been described. Even to the most casual reader it must have become evident that almost every case is a law unto itself, and is only surrounded by an atmosphere which stamps it as epilepsy. In some cases only a tonic or a clonic spasm occurs, or in others the mild symptoms of petit mal are present, of which the writer will speak further on. In the opinion of the author, as a rule, it may be laid down as a positive fact that when the fit is one of the tonic type it is generally of a less severe character than when it is clonic; but exceptions to this rule may occur, as is evidenced by the tetanoid epilepsy of Pritchard, in which rigidity of a most persistent and dangerous type often is present. However, it is undoubtedly true that attacks either entirely clonic or tonic are less severe than those which have both varieties of muscular disturbance.

After-Symptoms.—The heavy sleep or coma which follows immediately upon the retreat of the convulsion has been already spoken of, as well as the headache which follows the coma, particularly if the sleep be disturbed by the attendants of the patient.

[1] Quoted by Sieveking, *loc. cit.*

Headache.—The following table, showing the frequency of the occurrence of headache may be of interest[1] :—

Headache.	Males.		Females.		Total.	Per Cent.
Absent, ...	7		1		8	23.5
Present occasionally,[1] .	6	11	11	15	17	76.4
Present frequently, . .	5		4		9	
	18		16		34	

This shows very well the constancy of headache as an after-symptom, but in the writer's experience a much larger portion suffer from cephalalgia. Thus, in the cases of epilepsy collected by the writer, it was found that headache was recorded as present in 432 instances, and as absent in only 34 cases. In the remaining cases no record of the presence of headache was found, often owing to the carelessness of the person making the report. It will also be seen that females suffer from headache more commonly than males in the proportion of 93 to 61 per cent.

Coma and Sleep.—It has been held by some that the after-symptoms of sleep and coma are identical, and, while the point is well drawn, we cannot help evading it, simply because in some cases the condition is neither one nor the other. Thus, sleep is the state of unconsciousness commonly found in every one, but from which they can be more or less readily aroused if desired, but coma is a state in which no external influences are able to rouse the man from his lethargy. In epileptics either one of these conditions may assert itself.

The true relationship of this after-condition to the paroxysm is also a matter of dispute, some claiming that it is part of the seizure, while others think it only the natural reaction after the strain of the convulsion.

[1] Reynolds on Epilepsy. London.

The writer believes that a medium view is to be taken of this, and that both factors are at work, the exhaustion being probably the chief cause of the sleep. On the other hand, Siemens[1] contends very strenuously that this is not so, believing that the coma forms a third stage of the attack.

Paralysis.—Motor paralysis with loss of all power may succeed epileptic paroxysms, and this is particularly the case in those instances where the convulsive movements are largely unilateral in character. This condition has received the name from some neurologists of "post-convulsive paralysis" or "hemiplegia epileptica," and the frequency of its occurrence has been noted by Addison,[2] whose results are given in the following table.

In 31 epileptic males and 10 females paralysis was an epileptic sequel, as follows:—

	Males.	Females.	Total.
Paralysis of right side, . . .	9	2	11
Paralysis of left side, ...	7	2	9
Paralysis on both sides, . . .	1	0	1
Not paralyzed, ...	14	6	20

It will thus be seen that in 21 cases hemiplegia epileptica followed, while in 20 cases it did not occur,—a percentage which, if relied on, gives about 50 post-convulsive palsies to the hundred epilepties. In these cases the epileptic movements were both unilateral and bilateral, and the paralysis when unilateral was always on the side in which the fit commenced. The question at once arises, when considering this condition, as to the cause of the paralysis; or, in other words, is this temporary loss of power the result of exhaustion of the nervous protoplasm by the forcible discharges which it

[1] Allgem. Zeitschrift f. Psychiatrie, Bd. XXXV, Heft 5.
[2] Jour. of Mental Science, vol. xii, 1867.

has put forth, or does it arise from a condition of the nervous matter closely allied to actual organic change? That the loss is purely functional seems certain, for if it were organic the palsy would be permanent, not temporary as it is. Of course, where the epileptic attack is the result of an embolism, or is accompanied by an aneurismal dilatation of a blood-vessel, or an apoplexy, then the paralysis is organic; but in the simple fleeting paralysis after epilepsy no such change, of course, occurs. Gowers[1] expresses the belief that in severe fits the loss of power is purely functional and due to exhaustion, while that occurring in less severe attacks is due to an "inhibition of the motor centres," whatever that may be. So far as the writer is aware, centres possessing an inhibitory influence over the motor cells of the cerebral cortex are yet to be found to exist, and, while every one knows of their existence in the medulla oblongata, and probably in the spinal cord, no one has been bold enough to attempt to prove that they possess any power except over reflex phenomena. Dr. Gowers may express his idea in an unfortunate manner, but, as we understand him, his explanation is of a most hypothetical character and entirely without foundation either physiologically or otherwise, for he brings forward no support of it save arbitrary assertion, of which he is very fond. The majority of all neurologists believe the palsy to be the result of exhaustion, and find no necessity to confuse matters by advancing additional hypotheses concerning which they know nothing, and which have loaded down the boat carrying our knowledge of epilepsy in the past until it nearly sank.

Studies have been made by Féré[2] on the condition

[1] *Loc. cit.*, p. 688.

[2] Compt rendus de la Société de Biolog., Feb., 1888.

of muscular power of epileptics, both during the intervals between the attacks and immediately after the paroxysms. Using the ordinary dynamometer he found the general strength of such persons to be from one-third to one-fourth less than is normal. In his studies of the post-paroxysmal state he finds the strength always markedly decreased, and notes that this is more severe after nocturnal than diurnal attacks. This is not to be explained by any variation in his experiments, and seems to be as yet not clearly understood by any one.

The condition of the reflexes after an attack of epilepsy is one of much interest and worthy of special study. Ordinarily for the first few moments after an attack all the reflexes are lost, but immediately after this they are all very much increased and sometimes become so excessive that "ankle-clonus" may be readily elicited.

Reflexes.—A careful study of the condition of the various reflexes after an epileptic paroxysm has been carried out by Beevor,[1] who divides his cases into two classes, namely, those seen instantly after the paroxysm and those in which some moments elapsed before he began his tests. In all, he made observations on seventy fits occurring in 31 different cases, and examined both legs in each. His results are best given in a tabulated form:—

	Cases Seen Instantly.	Cases Seen Shortly.	Total.
Knee-jerk increased and clonus present, .	27	11	38
" diminished and clonus absent, .	6	7	13
" absent and clonus absent, . .	3	8	11
" normal and clonus absent, . .	4	1	5
" diminished and clonus present, .	2	0	2
" normal and clonus present, . .	1	0	1
	43	27	70

[1] Brain, April, 1882.

He also found that the plantar reflex was absent instantly after the clonic stage, but returned in from three to thirteen minutes, the average lapse of time before its return being five minutes. In every instance the plantar reflex occurred synchronously with the disappearance of the clonus, and in every case examined the attacks were bilateral, though in several they were a little more marked primarily on one side than on the other, and in these the knee-jerk and ankle-clonus were more marked on the side first affected. He also records a study of conjugate deviation of the eyes in the same class of cases, which, as it bears upon the results of the research just quoted, should be given here before considering their practical bearing.

Having first called to mind the fact that in most epilepties the fit begins by the rotation of the eyes and head to one side, generally the right, accompanied by flexion of the elbow and wrist of the same side, he goes on to describe the after-movements of the eyes when the fit has passed off, as the writer has done when considering the after-symptoms of the paroxysms, namely, the rolling of the eyes to the opposite side and their fixation there for some moments, followed by a rolling of the eyes from side to side like a very much exaggerated nystagmus. In his studies this occurred in 11 cases out of 13.

The argument by which practical gain can be reached by such cases is as follows: It will be remembered that in ordinary paralysis, as, for example, hemiplegia, we have exaggerated knee-jerk and ankle-clonus, and we also know that after epileptic fits we may have paralysis present of a transient or permanent character; but we are unable to prove its existence in every case because, with a return of a sufficient degree of consciousness, the

palsy passes away. In the one case the palsy is due to some injury of the nervous protoplasm; in epilepsy it is due most frequently, according to most clinicians, to exhaustion of the nerve-cells. The results are increased knee-jerk and ankle-clonus in either case, and this is why Beevor reached the results already given. The deviation of the eyes also supports this view, in all probability, because the eyes roll over to the opposite side from that most severely affected as a result of exhaustion or palsy of the centres governing them on that side, which, from its primary excessive action, has been more depressed than its neighboring centres on the other side of the brain. In other words, it would seem probable that, by noting the limb in which clonus and knee-jerk are most marked, and the side to which the eyes are secondarily turned, we can decide which side of the brain is diseased, particularly if the first movements of the fit are also noted, even in the cases where the paroxysms are most widely and bilaterally distributed.

As a proof of the accuracy of these arguments, a case may be detailed to illustrate them: A man was convulsed on the right side (a unilateral attack) for twenty minutes, and afterward, on this same side, there was always diminished plantar reflex and increased knee-jerk and clonus; while on the left or unaffected side there was scarcely any increase in clonus or knee-jerk and return of plantar reflex in three minutes, but on the right leg it did not return for thirty minutes. There was also palsy of the right side for two hours after each attack, with some aphasia.

The researches of Oliver[1] are also in accord with those of Beevor, for he finds that knee-jerk is sometimes present, sometimes absent, and foot-clonus is always present.

[1] Edin. Med. Journ., 1886, p. 211.

Bodily Temperature.—Some difference of opinion has existed as to the effect which the epileptic paroxysm has upon bodily temperature. A large number of cases collected and examined by Bourneville, in 1870, showed that there is always a slight rise of temperature, sometimes amounting to 2.5 ° Fahrenheit scale. Still more recently, the same investigator has carried his studies further on 82 cases, and reached identical results with his first conclusions.[1] He saw 2 cases where a fatal termination occurred, the temperature being 41.4 ° C. and 42.4 ° C.

Williams[2] also found that the temperature always rose—sometimes as much as 3 ° F.—after a fit.

Witkowski,[3] on the other hand, claimed, from a series of cases examined by him, that no change took place; and Westphal[4] thinks that elevation of temperature is rare, but he has been proved incorrect by the more recent studies of M. Bourneville and M. Georges Lemoine,[5] who examined no less than 200 cases of isolated attacks, with the result of confirming the earlier researches of Bourneville. It should be remembered, too, that this earlier observer found that the status epilepticus was capable of raising the temperature of the body to as much as 105 ° to 107 ° F.

Lemoine's results are perfectly in accord with those of Bourneville and with physiological reasoning, for he found that the temperature rises during the attack, but falls rapidly afterward, so that in a quarter of an hour it may have reached an almost subnormal point, but after two hours is found to be normal and to remain so

[1] Le Progrès Médical, No. 35, 1887.
[2] Medical Times, vol. ii, 1867.
[3] Berliner klin. Wochenschrift, Nos. 43 and 44, 1886.
[4] Archiv f. Psychiatrie und Nervenkrankheiten, vol. i.
[5] Le Progrès Médical, February 4, 1888.

constantly. In only one case—that of an epileptic idiot, with very violent attaeks, and, as a rule, subnormal temperature—was there any exception to this rule. The average rise, as found by Lemoine, is about 1.2 ° F., and the reetal temperature very rarely goes above 102 ° F.

The temperature in cases of epileptie insanity showed eonsiderable variation, even when there were no attaeks, and so did the temperature preceding different attacks vary eonsiderably; so that it was somewhat diffieult to attain a standard for comparison. The fall after the first quarter of an hour was very marked,—sometimes as mueh as 1.4° F. in that time,—and this occurred even after the most violent attacks.

In elose relationship with these studies are those of Addison,[1] undertaken with the view of discovering whether the temperature of an epileptic varied from the normal between the attaeks. Taking the temperature in the reetum in 13 men, he found the highest temperature to be 100.2° F. and the lowest 97.2° F., or a mean of 98.9° F., which is virtually just normal. He noted that the highest temperature oecurred in a man who had had an attaek that day, while the lowest was in an idiot.

Reynolds[2] has, by a series of investigations, shown that in 60.4 per eent. of the eases of epilepsy seen by him the temperature was normal between the paroxysms.

Aberrant Symptoms.—In some epilepties the eonjunetivæ and the skin of the faee are nearly always, after an attack of epilepsy, covered by small, hæmorrhagic points, due to the rupture of small blood-vessels. Closely allied to this is an interparoxysmal symptom that Sandras[3] has recorded a "*disposition singulaire et inex-*

[1] Journal Ment. Science, vol. xii, 1867.
[2] Epilepsy, London.
[3] Traité pratique des malad. nerveux, t. i, p. 203.

plicable" in the skin of epileptics, for when exposed to the sun they become covered on the face and even elsewhere on the body with numerous *taches rosée* without any elevation, and which disappear rapidly when the person goes into a shady spot. The writer has never heard of similar cases, and gives this abstract for what it is worth.

In others vomiting comes on before the coma has entirely passed away, and may produce death by suffocation, owing to the impaction of a piece of food in the larynx. Hunger is always present in the sthenic cases when recovery has gone on far enough for it to be felt, but in many of the weaker cases it is wanting, and is often replaced by anorexia. Between the paroxysms, unless the patient's stomach be disordered by drugs, the appetite is generally voracious.

Urine.—The rise of temperature, which has just been considered, leads us to the question as to whether there is, with the rise, a concomitant degree of tissue-waste evidenced by increased solids in the urine, and this point, unfortunately, is still undecided, for, although a good deal of work has been done, there are many contradictory results.

Some observers state that there are no changes in the amount of solids in the urine whatever, but it seems almost incredible that so much violent nervous and muscular exertion can take place without being accompanied by an increase in the phosphates, urea, and uric acid.

A very thorough study of this character has been carried out by Addison,[1] who analyzed the urine of patients for three days during and after the paroxysmal period, with the following result:—

[1] Journal Ment. Science, vol. xii, 1867.

CASE I.—*Paroxysmal Period.*

DAY.	Quant. of Urine.	Specific Gravity.	ClNa.	Urea.	PO_5.	SO_3.
	Ounces.					
First, . .	14.5	1033	21.14	325.64	14.44	28.61
Second, . .	17.0	1031	44.62	374.35	18.16	24.68
Third, . .	16.5	1034	43.41	375.37	26.27	43.67
Total, . .	48.0	1032	109.17	1075.36	58.87	93.96

Interparoxysmal Period.

First, . .	93.5	1016	54.54	572.68	29.60	29.38
Second, . .	99.5	1014	35.11	418.70	31.50	29.78
Third, . .	89.0	1013	64.89	414.00	31.15	26.64
Total, . .	282.0	1014	154.52	1405.48	92.25	85.80

CASE II.—*Paroxysmal Period.*

DAY.	Quant. of Urine.	Specific Gravity.	ClNa.	Urea.	PO_5.	SO_3.
	Ounces.					
First, . .	31.0	1021	67.81	379.75	22.74	21.76
Second, . .	26.5	1030	54.10	456.02	26.07	23.80
Third, . .	40.5	1025	59.06	472.50	10.70	26.68
Total, . .	98.0	1025	189.97	1308.27	59.51	72.44

Interparoxysmal Period.

First, . .	95.0	1017	60.59	881.19	152.39	45.51
Second, . .	91.5	1014	32.58	800.62	226.84	27.39
Third, . .	85.0	1015	40.40	614.83	111.56	34.36
Total, . .	271.5	1015	133.57	2296.64	490.79	107.26

CASE III.—*Paroxysmal Period.*

DAY.	Quant. of Urine.	Specific Gravity.	ClNa.	Urea.	PO_5.	SO_3.
	Ounces.					
First, . .	22.0	1030	77.00	385.00	31.18	23.00
Second, . .	7.0	1034	21.41	136.79	8.17	11.00
Third, . .	7.0	1030	20.41	147.00	5.37	7.85
Total, . .	36.0	1031	117.82	668.79	44.72	41.85

Interparoxysmal Period.

First, . .	35.0	1012	20.41	240.20	22.00	7.85
Second, . .	73.5	1006	42.87	192.93	16.21	9.80
Third, . .	51.0	1012	29.48	267.75	21.25	15.87
Total, . .	159.5	1010	92.76	700.88	59.46	33.52

To summarize, we find in these tables that in every instance the quantity of the urine was many times more between the paroxysms than during them, that the specific gravity was less at this time than during the seizure period, and that the amount of the chlorides was less in the intervals of repose than in the intervals of attack, except in the first case, where this is completely reversed by an unexplainable result.

The urea was considerably increased in amount during the interparoxysmal periods in every case, as were also the phosphates, while on the other hand sulphates were decreased, except in case No. 2, where they were increased during this period of quietness.

It is worthy of note that the urine was not only increased, during the quiet intervals, over the periods of attack, but that it was increased far beyond the normal amount, and, naturally, the specific gravity fell. It is equally worthy of remark that the chlorides grew less

at this time, and that the quantity of urea excreted was not greatest at the time of the greatest muscular action, namely, the time during the attacks. The elimination of phosphates, the representatives of nervous change, was similarly affected. Echeverria[1] found the amount of urea excreted after a fit to be much greater than the normal, but Gibson[2] found no change in the urine.

In some researches made by Beale[3] the same results were reached, but were arrived at somewhat differently. On analyzing the urine of four persons suffering from epilepsy, he found that the phosphates were always increased by the fits, particularly if these attacks were frequently repeated or very violent.

CASE I.—*Urine: Specific Gravity, 1.024.*

Water,	931.2	
Solids,	68.8	100.
Organic matter,	58.35	86.27
Fixed salts,	9.45	13.73
Phosphates precipitated by chloride of calcium and ammonium,	6.96	10.11

CASE II.—*Urine: Specific Gravity, 1.024.*

Water,	927.2	
Solids,	72.80	100.
Organic matter,	51.01	85.18
Fixed salts,	10.79	14.82
Phosphates precipitated by chloride of calcium and ammonium,	3.92	5.38

CASE III.—*Urine: Specific Gravity, 1.017.*

Water,	958.8	
Solids,	41.20	100.
Organic matter,	34.68	84.18
Fixed salts,	6.52	15.82
Phosphates precipitated by chloride of calcium or ammonium,	2.15	5.21

[1] Epilepsy, p. 288.
[2] Medico-Chirurgical Transactions, p. 75, 1867.
[3] British Medical Journal, November 26, 1859.

CASE IV.—*Urine: Specific Gravity, 1.009.*

Water,	976.7	
Solids,	23.30	100.
Organic matter,	17.46	75.94
Fixed salts,	5.84	25.06
Phosphates precipitated by chloride of calcium or ammonium,	1.79	7.68

Hamilton[1] states that the urine of epileptics is apt to contain evidence of tissue-waste, and an increase in the amount of earthy phosphates as well. Zapolsky found, however, that immediately after the attack there was a diminution in the quantity of the phosphates, thus agreeing with Addison. De Renzi[2] has also published an article confirmatory of these results.

A research arranged in every detail to avoid fallacy is sadly needed.

Albuminuria and Diabetes.—Some persons have claimed that albumen is constantly present in the urine of epileptics, but they are incorrect, although there are researches which contradict this last assertion.

Saundby,[3] who tested the urine twenty-seven times in 20 cases, with the object of detecting albumen, using the boiling and nitric-acid tests, found it present on twenty-two occasions, as did also Otto,[4] who found albumen in the urine in 22 cases out of 31 epileptics. Indeed, Otto thinks albuminuria to be symptomatic of epilepsy. On the contrary, Mabille,[5] in a series of experiments, most carefully carried out on 38 cases of the disease, 20 of whom were men and 18 women, found not a trace of albumen in the urine of any one of them, although he tested for it before, during, and after the

[1] *Loc. cit.*
[2] Gior. internaz. d. Sc. Med., Napoli, ii, 357, 1880.
[3] Medical Times and Gazette, October 14, 1882.
[4] Berliner klinische Wochenschrift, October 16, 1876.
[5] Annales Méd. Psycholog., November, 1880.

paroxysm. In 1 case, which was known to have parenchymatous nephritis, it was found. Dowse[1] also has confirmed Mabille in this, but Huppert[2] agrees with Saundby and Otto that albumen is commonly found. When we consider the care used by Mabille and the possible carelessness of the others, and associate with this the fact that Karrer[3] has denied the presence of albuminuria after testing again and again by all known tests the urine of 12 chronic epileptics, and that Kleudgen[4] and Bunzlau agree with him, after a separate research, in supporting Mabille and Dowse, one cannot fail to be impressed, we think, by the fact that the condition of albuminuria in epilepsy is rather a chance occurrence than a regular concomitant.[5] In the tests which the writer has made of the urine of epileptics he has never found albumen.

Sugar is probably no more frequently found in the urine of epileptics than it is in that of other persons, for Addison[6] has tested the urine of 20 cases with negative results.

Bodily Weight.—Closely associated with the question of tissue-change in this disease is the assertion made by Kowalewski,[7] that every fit is accompanied by a loss of bodily weight ranging from one to twelve pounds, which, if true, shows that, either directly or indirectly, great changes in nutrition must occur. On the other hand, Lehman, Beevor,[8] Jolly,[9] Hammond,[10] Olderogge,[11]

[1] Practitioner, October, 1878. [2] Arch. f. Psychiatrie, p. 189, 1877.

[3] Berl. klin. Woch., July 5, 1875. [4] Arch. f. Psychol., Bd. XI, Heft 2.

[5] For additional facts reached by other observers, see Brüninghausen, Allg. Med. Central. Zeitung, Berlin, 1880, xlix, p. 97; and Fiori, Osservatore, Torino, 1880, xvi, p. 177; and Italia Medica, Genoa, 1881.

[6] Journal Nervous and Mental Disease, vol. xii, 1867.

[7] Arch. f. Psychiat., Bd. XI, Heft 2. [8] Brit. Med. Jr., July 8, 1882.

[9] Berliner klin. Woehensehrift, November 26, 1881.

[10] Journal of Nervous and Mental Diseases, p. 517, 1880.

[11] Archiv f. Psychiatrie, Bd. XII, Heft 3.

Kranz, and Schuchard assert equally positively that no such change occurs, and adduce such an array of experimental evidence that they cannot be mistaken. The method of research carried out by the Englishman, Beevor, consisted in having the patients weighed each morning at the same hour and under the same conditions in order to form a standard scale of weight. As soon as an attack had passed off they were weighed again to discover if any difference had taken place between the normal weight and that after the attack.

In 25 cases no decrease was noted; in 1 it was found that there was an increase in weight, but this was evidently owing to the fact that the patient had just had dinner. In 6 cases of hystero-epilepsy there was no change. In the 30 cases examined by Lehman the results were perfectly in accord with those of Beevor, and may be considered as even more accurate in that on several occasions he managed to get the weight of a patient immediately before and immediately after the paroxysm. Jolly's observations were apparently carefully made, and included 28 cases, while those of Hammond were made on 6 epileptic patients. Without intending to deny the truth of Beevor's studies, it must be remembered that a great element of fallacy was present in them all. He does not state whether the morning weighings were before or after breakfast; so that the nourishment taken, if the weighing was before breakfast, might have counterbalanced the loss by reason of a fit. Further, while he recognizes the influence which meals may have on his results, he failed to take any weights in the afternoon for his normal standard scale, and it is manifestly incorrect to say that a morning weight can be taken as a standard for the entire day.

This question has also been studied in a somewhat novel manner by Hallager,[1] of Viborg, and, though he agrees largely with Kowalewski, some of his results are interesting as pointing toward a possible solution of the controversy.

After a large number of studies he has, by means of a chart, shown that whenever there is a loss of weight there is an increase in the amount of urine voided by the patient. In other words, as the weight-curve falls the urine-curve rises. He deduces from this that the loss of weight, sometimes seen, is by loss of liquid from the body, and that the lessening of weight is not, in reality, due to tissue break-down. That it is certainly not the latter is proved by the fact that nutrition is usually remarkably well preserved in epileptics. Reynolds[2] finds it impaired in only 12.3 per cent., and that the strength only fails in 24.4 per cent. That Hallager may be correct in his explanation seems likely by the measurements of urine made by Addison (*loc. cit.*).

Sensory Disturbance.—The amount of disturbance of the sensory apparatus after epileptic attacks has been studied within the last few years most carefully by several observers, notably Thomsen,[3] who announced that cutaneous and sensory anæsthesias often exist in epilepties permanently, and in connection with Oppenheim,[4] in 1884, he published an elaborate essay on the subject. These investigators found that an examination of 94 cases of epilepsy showed that no sensory disturbance acutely follows an epileptic convulsion, but that in old epilepties, with more or less deficiency of cerebral power, sensibility was greatly diminished. There are

[1] Nordisk. Med. Arkiv, 1886.
[2] *Loc. cit.*
[3] Neurologische Centralblatt, xxiii.
[4] Archiv für Psychiatrie, xv., p. 558.

exceptions, however, to the rule that no sensory disturbances follow acute attacks, for they found that anæsthesia of a temporary form was present whenever the convulsion was slightly aberrant in type, and, while truly epileptic, accompanied by rare symptoms. They divide such cases into three divisions as follows:—

1. Those in which the convulsion is followed by delirium and hallucinations. 2. Those in which the attack is followed by emotional or psychical phenomena of a severe form, with delirium. 3. Those in which the attack seemed to expend itself chiefly on the mental portion of the organism.

It is at once evident, however, that these attacks are hysteroid in form, and in reality very irregular. On the other hand, it is perfectly possible that the epileptic storm may exhaust the higher sensory centres *pari passu* with the depression of the motor area. The great difficulty, too, of obtaining satisfactory answers from many epilepties, for some time after an attack, surrounds all such researches with some doubt.

Aphasia.—A condition which may be present after epilepsy, not uncommonly, is *aphasia*, due, in all probability, where it is temporary, to collateral exhaustion of the gray matter containing, not the centre of speech, but those centres for the muscles which practically form the words as spoken.

The Eye.—It is interesting to know that the eye-grounds of a very large number of persons have been examined, in order to determine whether any great changes take place in persons who are epileptic. Cross[1] has examined the intra-ocular circulation of 95 cases, and found that in the great majority no change was noticeable. He also finds that no structural changes in

[1] Jour. Nervous and Mental Diseases.

the eye take place. In the cases where any changes were noted in the intra-ocular circulation, the results were so various and widely dissimilar that no conclusions could be drawn. Gowers[1] states, as a result of an examination of over 1000 eye-grounds in epileptics, that no changes at all are to be seen in the fundus oculi of idiopathic epilepsy. He states, however, that the retinal arteries have seemed unduly large, while the veins have not, except during an attack, when the veins are distended. Allbutt, of England, has seen pallor of the optic disks, as have also Hughlings-Jackson and Aldridge.[2] Hammond[3] believes, in distinction from Gowers, that the fundus of the eye of an epileptic is always congested or pale, and evidences cerebral congestion or anæmia, and he sees a venous pulsation in all cases of plethora with epilepsy. Köstle and Niemetshek,[4] of Prague, from their examinations, state that the venous pulse of the fundus only occurs in those cases where there is anæmia of the brain, and they go so far in their theory that they believe all epileptic brains are anæmic. Tebaldi[5] has also recognized pallor of the papilla, increase in the venous circulation, and immediately after an attack marked congestion of the veins, with relative emptiness of the arteries. Abundo,[6] of Naples, has studied the eyes of this class of patients, apparently with great care, and finds that after a fit the ophthalmoscope reveals the vessels of the fundus congested according to the severity of the attack, but that no difference is to be seen in the two eyes. There is always contraction of the visual field in both eyes, but

[1] Medical Ophthalmoscopy, p. 157.
[2] West Riding Lunatic Asylum Reports, vol. i.
[3] Nervous Diseases.
[4] Prager Vierteljahrschrift, Heft cvi und cvii, 1870.
[5] Riv. Clin., ix, 1870.
[6] Ricerche Cliniche sui Disturbi Visivi vel l'Epilessia Napoli, 1885.

no hemianopsia, while the visual acuity is dimmed in direct ratio with the violence of the paroxysm.

Pupils.—During the coma and sleep following the convulsion the pupils contract more sluggishly to light than is normal, and if the mental condition of the patient is one of depression the pupils are found tightly contracted after the fit. Abundo denies, however, that rapid changes occur in the pupil after an attack; but in this he is mistaken, at least in respect to some cases. Again, he finds no proof of Siemen's assertion that dilatation of the pupil, at the beginning of the fit, is first preceded by contraction.

In 1880 Dr. Gray,[1] at a meeting of the American Neurological Association, read a paper in which he insisted, very strenuously, that the pupils of epilepties were more widely dilated at all times than in normal individuals, and that they were more mobile than those of healthy persons or persons suffering from other convulsive disorders. Indeed, he asserted that this was a pathognomonic sign of epilepsy to be used in making a differential diagnosis. In the discussion which followed, Spitzka[2] pointed out that Gray was mistaken, because the dilatation spoken of was replaced by contraction in the epilepsy of alcoholism. Dr. Cross[3] and Dr. Hammond[4] supported Gray, not by means of any observations, but simply on the basis of their impressions as derived in practice. Since that time, however, careful observers on the other side of the Atlantic have proved, by the most painstaking observations, that Gray's statements are entirely lacking in fact.

Marie,[5] at the request of Charcot, in 1882 studied the subject accurately, placing a candle distant from

[1] Jour. of Nerv. and Ment. Dis., 1880. [2] *Ibid.* [3] *Ibid.* [4] *Ibid.*
[5] Arch. de Neurolog., vol. iii, p. 42, 1882.

each eye 45 centimetres, the rays of light falling on the cornea at an angle of 45 degrees to the visual axis. Hutchinson's pupillometer was used, and studies made on 53 epileptic women and 10 healthy nurses. In the epileptic patients the diameter of the pupil was $5\frac{1}{2}$, in the nurse $6\frac{1}{3}$, which is of course just the reverse of Gray's statements. Marie also found the pupil no more mobile in epilepties than in others.

Musso,[1] who made similar tests with 70 epileptics and 10 healthy persons, reached conclusions corresponding to those of Marie, and states that the pupils of epilepties show no difference from those of normal persons. He found, however, that there was in 22.8 per cent. an inequality of the pupils.

Refractive Anomalies.—Those who have read the interesting essay of Dr. G. T. Stevens on "Functional Nervous Diseases," which was honored by l'Académie Royale de Médecine de Belgique at the *concours* for 1881–83, must have been struck by the statistics which he gives as to the ocular conditions found in 140 cases of epilepsy, 85 of which were in private practice. To use his own words, "The general results of these examinations has been to reveal the existence of refractive anomalies in a considerably greater proportion than has been found by Cohn in his examinations of the eyes of school-children, or by other observers in similar investigations prosecuted in Germany, Russia, and America." Dr. Stevens found that in 100 consecutive cases there existed :—

Hypermetropia (including hypermetropic astigmatism) in	59
Myopia (including myopic astigmatism) in . . .	23
Emmetropia, or refractive errors less than one dioptric, .	18
	100

[1] Riv. Sperimentale di Firenze, fasc. i and ii, 1884.

There is one criticism which may be made in regard to these statistics of Dr. Stevens, namely, that the percentage of hypermetropia is not sufficiently over and above the normal percentage, as found by Cohn, to be of any value in pointing to a greater frequency of this error in epilepsy than elsewhere. Furthermore, the writer does not believe that 100 cases give a wide enough experience to justify any one in attempting to formulate general rules in regard to such a disease as epilepsy. The very large amount of work done by Cohn on persons presumably healthy as to their general condition should be offset by an equally large series of pathological examinations before conclusions are developed.

Dr. Stevens evidently sees that such a criticism may be made, but does not allow it to alter his reasoning.

In the same essay the fact is pointed out that muscular insufficiencies in the ocular apparatus are potent factors in the development of epilepsy, and a very remarkable array of cases is brought forward to show the correctness of this view. The truth of this as applied to the treatment of epilepsy in the hands of other members of the profession has yet to be adduced, but no evidence exists, so far as the author is aware, to rebut it.

Mental State.—A very important question connected not only with the prognosis of epilepsy but also with its relation to medical jurisprudence lies in the influence which the disease may exercise on the mental condition of the sufferer. Russell Reynolds,[1] of London, who has written very extensively on this subject, has arrived at the following conclusions in regard to the effects of the disease on the intellect, and, when we remember that

[1] Epilepsy, its Symptoms, Treatment, and Relation to Other Convulsive Disorders. London, 1862.

some of the greatest men that ever lived[1] were afflicted with epilepsy, it is not difficult to agree with him, although his conclusions cannot be accepted without some qualifications. He believes :—

1. That epilepsy does not necessarily involve any mental change.

2. That great mental impairment exists in some cases, but this is the exception rather than the rule.

3. That females suffer (in mental vigor) more commonly than males, and also more severely.

4. That the commonest failure is loss of memory, and that this, if regarded in all degrees, is more frequent than integrity of that faculty.

5. The apprehension is more frequently preserved than lost.

6. That ulterior mental changes are rare.

7. That depression of spirits are common in males, rare in females, but that excitability of temper is found in both sexes.

If we think over these conclusions we are struck with the nicety of the line drawn by Reynolds between mental change and other conditions which we are accustomed to associate in our minds with normal mental processes. Of his first conclusions we shall speak in one moment, but at present we shall consider only the fourth. In the fourth he states that loss of memory is more frequently present than retention of it, and one cannot but think that in this he contradicts himself in that memory is certainly a function of the brain, and its loss is universally associated with impairment of its functions.

It seems certain that his first statement cannot be

[1] Napoleon Bonaparte was undoubtedly epileptic, and it is stated that he had a seizure whenever he had sexual intercourse. Cæsar was also a sufferer from the disease.

denied, for some cases do go on having epileptic paroxysms for long periods of time without mental involvement, as, for example, Cæsar and Napoleon; but his second conclusion is certainly open to criticism. The writer is quite confident that the facts are just the reverse, and that impairment of mental power is the rule rather than the exception. As to his fifth proposition, in which he states that apprehension—by which the writer supposes he means the power to grasp an idea—is more frequently preserved than lost, it is almost impossible to offer evidence, since this power varies so in persons classed among us as healthy; but in the sixth, which is closely related with the first, the writer believes he is sadly at fault. He thinks, too, that the seventh and last conclusion supports what he has already said in respect to the fourth, namely, that some mental change does generally occur. Not only are the remarks just made true, but it is positive that all the more modern writers utterly disregard all the deductions drawn by Reynolds. Notable, among these, we find Falret,[1] in France, and no less an authority than the famed Charcot,[2] while in Germany, England, and America the voice of the most prominent neurologists are raised in the support of this belief.

Morel[3] has in his writings called attention to these changes, and has defined the mental condition of epileptics as the "epileptic character." Hasse[4] has likewise insisted that mental change occurs, and Romberg[5] says

[1] De l'État Mental des epileptiques, Arch. gén. de méd., Avril et October, 1861.

[2] Leçons sur les maladies du système nerveux, series ii. Paris, 1873-7.

[3] l'Études Clinique. Traité theorique et pratique des maladies mentale, tome ii, p. 316.

[4] Krankheiten des Nervenapparat. Virchow's Handbuch f. Spec. Path. und Ther.

[5] Handbuch f. Nervenkrankheiten, vol. ii.

it is charaeteristie to find a loss of memory and diminution of the distinctness of ideas, eombined with great irritability of temper. Esquirol [1] stated that four-fifths of all the epileptic women in the Salpêtrière were insane, and that the remaining one-fifth were singular in their conduct, while Foville[2] writes that mental failure oecurs in the majority of eases. Georget[3] also writes that the disease terminates in mental deterioration. On the other hand, Maissonneuve[4] details a ease of severe epilepsy in which the attaeks were frequent, but in whieh no evidence of mental failure existed. Of eonrse, we all know that these eases do occur, but they are rarely to be found as eompared to the others.

Gowers[5] states that the interparoxysmal mental state of epilepties often presents grave deterioration, and that this is one of the most serious, and most dreaded, effeets of the disease. In the beginning there is merely defective memory, but later the intelleet generally suffers and there is often defective moral control. In some instanees, he states that actual imbecility may be reached. In America we find Hamilton[6] stating that mental decay is frequent and an ultimate resnlt, in which mental enfeeblement, with progressive and great loss of memory, ending in total dementia, by no means rarely oeeurs. Wood[7] also speaks of it as a common sequel. The writer's own experience eertainly eonfirms the results given by the others just quoted.

As so mueh evidence has been offered against the

[1] Des maladies mentales eonsiderées sous les Rapport's médieal, hygienique et médieo-legal, tome i, p. 285.

[2] Dietion. de Méd. et de Clin. pratique, art. Epilepsie, p. 416.

[3] De la Physiolog. du système nerveux, tome ii, p. 385.

[4] Recherches et Observation, p. 58.

[5] *Loc. cit.*, p. 692.

[6] *Loc. cit.*, p. 482.

[7] *Loc. cit.*

validity of Reynolds's conclusions, it is, perhaps, but just that the writer should at least place before the reader the evidence by which Reynolds reached them. He divided his cases into four classes as follows :—

1. Those in whom no change in mental power was evident to the nearest friends and relatives of the patient, and of these there were 16 males and eight females, or a total of 24, or 38.70 per cent.

2. Those in whom the memory for recent events was partly clouded, but who had the memory for things long gone by well preserved, of which he found 10 males and 10 females, or a total of 20, or 32.25 per cent.

3. Those in whom there was diminution of apprehension and loss of memory. Four males and 5 females, or a total of 9, or 14.51 per cent.

4. Includes those possessing changes of the second and third class, and, in addition, evidences of great stupidity. Of these there were 4 males and 5 females, or a total of 9, or 14.51 per cent.

He also carried his examinations still farther, as regards the condition of memory alone, in distinction from general mental impairment, with the following results from 57 cases :—

Condition of Memory.	Males.	Females.	Total.	Per Cent.
Normal,	14	8	22	25 = 43.85
Defective only after fits,	2	1	3	
Slightly defective at times,	2	2	4	32 = 56.14
Defective generally,	9	9	18	
Very defective,	6	4	10	
	33	24	57	

In another table he studies the condition of "apprehension," as he terms it, or the power of grasping ideas :-

Condition of Apprehension.	Males.	Females.	Total.	Per Cent.
Normal, ...	17	12	29	30 = 62.5
Defective only after attacks,	1	0	1	
Periodically affected, . .	1	1	2	
Generally defective, . .	4	5	9	18 = 37.5
Extremely defective, . .	4	3	7	
	27	21	48	

It is worthy of note that these results show epileptics to be more frequently deficient in memory than in apprehension in the ratio of 56 to 37.

Epilepsy is also closely connected with other conditions of mental lack of power than that brought on by the disease itself, and in some cases the loss of intellect precedes the development of seizures. When this is not the case we frequently have transient mental disorders developing, which have been hinted at, and of which we shall speak in a moment. Ingels, superintendent physician of the Hospice-Grislain in Ghent, has, in a paper read in the International Congress of Psychiatry and Neuro-Pathology, in 1885, attempted to show the relations between epilepsy and idiocy. Out of 79 children under his care 25 were epileptics, and out of 398 children received into the institution in twenty-eight years 125 were epileptic idiots,—nearly one-third. These he divides into two classes: (1) those in whom epilepsy and idiocy were congenital and (2) those in whom epilepsy had caused dementia. He found that in the latter class the failure of mental power was always very rapid.

In some studies made by Howe [1] of 574 idiots, he found that 125 had epilepsy, but of these 92 were idiotic from birth and 33 had acquired idiocy, whether as a result of the epilepsy or not is not stated. Probably the idiocy was not the result of the epilepsy, for idiocy is

[1] On the Causes of Idiocy, p. 56. London, 1856

rarely a sequence of this disease, imbecility being generally the ultimate condition. The figures by Howe should not, therefore, be taken as statistics on the frequency of epilepsy and idioey, but rather of idiocy and epilepsy,—two entirely different things. An interesting fact in regard to epileptic idiots is that they seem to preserve some signs of brain-power, and to be able to learn simple things, which are, however, swept completely out of their minds by the first repetition of an attack.

Insanity, in distinction from idiocy or imbecility, rarely complicates epilepsy, although Bucknill and Tuke,[1] the two well-known English psychologists, state, from an analysis of many thousand cases, that the percentage of persons insane by reason of epilepsy is not less than 6.5 per cent. In the "Rapport sur le Service des Aliénés du Departement de la Seine," for 1877, the principal causes of insanity are given in 2068 cases, of which number 59 were due to epilepsy. In 864 cases, where the causes of insanity were discovered, admitted to the Lunatic Asylum at York, 23 were due to epilepsy, and in the 687 cases admitted to the Northampton Asylum, in the United States, 62 cases were supposed to be due to epilepsy. It should be remembered that insanity is rarely complicated by epilepsy unless dementia paralytica exists. Epileptic insanity has a most unpromising outlook, and the prognosis should always be most unfavorable. Cases of cure are almost unknown.

According to the studies of Echeverria,[2] nocturnal epilepsy is much more apt to be complicated by insanity than is diurnal epilepsy, and petit mal is more apt than either of the violent convulsive varieties to be followed

[1] Psychological Medicine. 4th ed.
[2] International Congress of Mental Medicine. Paris, 1878.

by mental overthrow. Hallucinations he also found to be very common in epileptic insanity, amounting to as much as 86 per cent. in 267 cases. The hallucinations were divided as follows: Auditory, 62 per cent.; sight and hearing, 42 per cent.; smell, 6 per cent.; while 70 per cent. showed anæsthesia, hyperæsthesia, or false sensations.

In the careful and interesting studies of Falret, he divides the mental disorders of epilepsy into three divisions, in the first of which are those in whom mental disease is between the attacks and independent of them; in the second, those in whom the mental derangement takes place during the paroxysm; and in the third class he places those in whom the intellectual disorder comes on in the attacks and lasts afterward.[1]

Echeverria also divides epileptic insanity into three classes, namely, into intermittent, remittent, and continuous, all of which terms designate the conditions so clearly as to require no explanation.

In the class of cases where mental disturbances are merely present at or about the time of the convulsion, we may have any variety of disordered function, all the way from pure idiocy to homicidal mania. In this we have a well-marked difference from the mental disorder produced by long-standing epilepsy, which is well to remember, for in the latter the alienation is one of imbecility and harmlessness, while in the first it may be quite the opposite. In some individuals there is a very early tendency to the development of mania; there is a certain periodicity about the explosions, and, when established, the excitement either precedes the attack by a few days or occurs shortly afterward. The violence is

[1] I have not space in which to quote this interesting research further, but to those who are interested I would refer them to the original.

eharaeteristieally aeute, and the mania is, while often homieidal, rarely suieidal. Fortunately, this mania only lasts, as a general rule, a few minutes or hours, but it may, as just stated, last mueh longer.

When it does last, it more frequently is assoeiated with hystero-epilepsy than epilepsy, and it is also worthy of note that in these eases the fits seem to produce some lessening of the mania immediately after eaeh one. That permanent mania is only sometimes seen in epilepsy is proved by the eases of Russell Reynolds (*loc. cit.*), in whieh he saw only 7 maniaes in 69 eases.

Responsibility of Epileptics.—The responsibility of sueh persons, so far as their aets make them amenable to the law, is a question which it is impossible for the writer to disenss here, simply beeause it hardly conеerns us in this essay, and because an enormous amount of legal as well as medieal writing must be eited, and deep questions into the delieate subjeet of the dividing line between responsibility and insanity are too devoid of results in the eourt-room to be of value either to the lawyer or to the physieian. There are many of us who are subjeet to harmless eeeentrieities whieh pass unnotieed in daily life, but which, if they were productive of more dangerous results, might very generally be regarded as evidenees of insanity or deranged mental aets.

Further than this, as epilepsy and insanity go hand-in-hand, without any relationship to one another, save that they oeeur synehronously, in many eases a seeond niee point of differentiation arises as to whether a criminal act results from an epileptic homieidal explosion of gray matter, to an insane homieidal aet, or to revenge aetnated by a real but insuffieient ause. It is just here that the physician finds himself, when eross-questioned,

unable to give any opinion which cannot be attacked. A few cases which are appended illustrate these difficulties very well, and in the present state of our knowledge render it impossible for any one to elucidate them, or to accurately judge as to penalties to be inflicted. Thus, in a certain case of epilepsy recorded by Thorne,[1] there were periods in which the ordinary fits seemed replaced by mental storms. Though usually a mild and good-tempered man, he would during these attacks seize a knife and declare he would kill his children. In another case, recorded by Orange,[2] a woman subject to epileptic seizures rose up from her chair with her baby in her arms, and began to cut some bread for an older child. While thus employed a fit came on, and she cut the hand of the baby right off, being found by the neighbors afterward in the coma following the fit. In the intervals this woman was entirely sane, but surely was not responsible for the injury to her child.

The following case recorded by Hamilton[3] is interesting in this connection :—

C. O., aged 22 years, a reporter attached to a daily newspaper, received an injury to his head when but 3 years old. He fell from the second story of an unfinished building to the cellar, striking the upper and back part of his head upon a beam. He was rendered unconscious, and remained so for a day or more. He recovered from the immediate bad effects, but suffered from severe general headaches, which recurred every week or so, with an increase in the amount of urine excreted. About six months before he was seen by the reporter he began to have epileptic convulsions of a violent character

[1] St. Bartholomew's Hospital Rep., 1870.
[2] Broadmoor Asylum Rep., 1877.
[3] Pepper's System of Medicine, vol. v.

almost every day, and sometimes more frequently. They were brought on by excitement, and he had a great many when worried about his wife at the time of her delivery. The attacks were, as a rule, preceded by an epigastric aura of long duration, and occasionally by a visual aura. At these times he was noted to be queer and strange; he would restlessly wander about his office, and suddenly, without any cry, become convulsed. After the attack he slept soundly. He often felt inclined to do himself an injury, or to kill some one. He was irritable, pugnacious, and he would often raise his hand to strike some one of his family and afterward know nothing of his conduct, and was greatly astonished when told that he had done so.

On one particular morning he was seen at 10 o'clock, having had a fit in his office at 8, of a more severe character than usual, and preceded by a psychical aura, during which he was very morose and sullen. Upon recovery he was speechless, though he could communicate by signs. He was sensible, but dazed, with his pupils strongly dilated, but mobile when the eyes were turned toward the light. When asked a question he understood it perfectly, and wrote an intelligible answer. He tried hard to speak, and expressed annoyance at his inability to do so. Laryngeal examination showed absolutely no cause there for his aphonia. He remained speechless all day. He was in bed later in the day, and able to speak one or two words with great difficulty. The grasp of the right hand was weaker than that of the left. He now expressed himself as tired, and, turning over, began to belch up wind; the muscles of the neck and right side became tonically contracted, the extensors predominating. He remained with the right arm and leg stiffly extended for a moment, then took two or three deep

inspirations, put his hand to his throat, and said that a bone had fallen from his throat. He now denied ever having lost his speech, and of seeing any of his physicians before, and he had forgotten all the previous occurrences of the day.

This, then, is another instance of the absolute irresponsibility of some epilepties during certain periods, and if murder had been committed the man should, undoubtedly, have been acquitted on the ground of temporary insanity.

Now, it is just such cases as these that lead the layman and the judges to a wrong impression. Many of my readers doubtless recall the famous case so often quoted by Tronsseau in his lectures, of a Parisian master-builder who was habitually seized with attacks in which, although entirely unconscious, he would run from scaffold to scaffold, springing from plank to plank, but never falling, and who certainly was absolutely irresponsible for his actions during that time. Again, the case of a Negro has been recorded by Wood, who would suddenly dash along the street uttering a scream, and would keep on as hard as he could run for from half a mile to a mile, when he would stop perfectly conscious, although entirely ignorant of the occurrences which had just taken place. If he were caught and held he was instantly thrown into a furious epileptic convulsion. If this man had committed a murder as he ran and been tried, no one, save the experts called, would have listened to the idea that he was irresponsible. These last two cases are, in reality, types of the disease described under the head of epilepsy procursiva, which has already been alluded to.

A very important point in this connection is the fact that whether a man be permanently insane or temporarily

suffering from homicidal mania, he equally requires constant watching. Because a man is sane for twenty-nine days in every month but homicidal on the thirtieth day is in reality a greater reason for confining him than one who by his constant aberration of mind warns those about him to be on their guard. The periodically epileptic homicide should be imprisoned, not after he has committed an outrage, but before he gets an opportunity.

Psychic Epilepsy.—A condition which is very intimately connected with what has been said on the last few pages is one which Weiss[1] has recognized and named "Psychic Epilepsy." Indeed, it is virtually identical with the case of C. O., which was given a moment since. In the "psychic epilepsy" of Weiss he recognizes a special form of the disease, running a typical course, and in which the convulsive attacks are replaced by psychoses, which stand in no relation to convulsions and have nothing to do with them. The mental disorder is characterized by a sudden onset, without incubation; it speedily, or in a few hours, reaches its acme, then rapidly disappears and is followed by complete psychic restoration and by a periodic recurrence without any failure of mental power. Weiss has seen 4 such cases, in all of which the course just given was closely pursued. In the first the man suffered from frequent attacks of insanity lasting two or three days, preceded by a well-marked aura. After the attack he was perfectly sane, but had absolutely no remembrance of the insane period. In the second case there was a history of vertigo, pain, and confusion of mind for thirty years previously. At this time spells came on associated with frantic runnings to

[1] Psychiatrische Studien aus der klin. von Prof. Leidesdorf. Wien, 1877. Centralbl. f. d. med. Wissenschaften, No. 15, 1877.

and fro, and accompanied by shoutings of the word "fire," which lasted from two to three days.

In the third case such attacks came on regularly two or three days before each menstrual epoch, accompanied by melancholia, which was followed by well-developed dementia, and finally a return to perfect health. The fourth instance illustrates what the writer has already said. A boy, generally perfectly well, was occasionally seized with a desire to kill something which he thought he saw, and which remained before him for but a minute or two. According to Weiss this boy ultimately became truly epileptic, and he believes that such a metamorphosis may frequently occur.

Syphilitic Epilepsy.—Syphilitic epilepsy is only one of the many nervous affections which afflict those who may be so unfortunate as to contract this disease. The manner in which the nervous outcome of the disease is reached will be considered under the head of Etiology. There can be no doubt that syphilis produces an enormous amount of epilepsy, and the presence of epilepsy in a person in whom the slightest chance of a specific taint exists should cause him to be instantly placed under antisyphilitic treatment. That this is true is evidenced by the statements of the best neurologists the world has ever known, for we find no less noted a man than Charcot[1] stating that epilepsy is the most frequent manifestation of cerebral syphilis, and the equally eminent Fournier,[2] the syphilographer, has insisted most strongly on this point, as have also Braivais[3] and M. Lagneau.[4] In England, Hughlings-Jackson, Broadbent,

[1] *Loc. cit.*

[2] De l'Epilepsie syphilitique tertiare. Leçons professie. Paris, 1876.

[3] Thèse de Paris, No. 18, tome iv, 1827.

[4] Maladies syphilitique du système nerveux. Paris, 1860.

Todd, and Buzzard[1] have promulgated this doctrine, and in America Weir Mitchell,[2] Spitzka, Wood,[3] and Carter Gray have recorded their belief in this idea, as have also Nothnagel and many equally eminent Germans. Indeed, it would be hard to discover any one statement in medicine which would receive more wide-spread assent on all sides than does this one.

The symptoms of syphilitic epilepsy really differ in no way from those of the simple idiopathic variety, but some questions peculiar to this form of the affection are well worthy of attention.

First and foremost it is exceedingly important to discover how long, after the syphilitic poison has been received, it is before the nervous outbreak results.

In some statistics collected by Echeverria[4] he found that in 118 cases of syphilis the first epileptic spasm was, in 65 males and 53 females, as follows:—

In	9	males	and	15	females	in	from 4 months	to	1	year.
"	16	"	"	20	"	"	1 year	to	2	years.
"	13	"	"	8	"	"	2 years	to	5	"
"	15	"	"	5	"	"	5 "	to	8	"
"	9	"	"	3	"	"	8 "	to	12	"
"	3	"	"	2	"	"	12 "	to	20	"

The ages of the males ranged from 19 to 30 years, while those of the females were from 21 to 28 years. On analyzing this table further than its originator did, we find that the average period after infection equals, as a general rule, from 2 to 5 years.

The previous symptoms to the epileptic attack were, in the 118 cases, as follow:—

Headache in 45 males and 38 females=83 patients=70.30 per cent. of them all.

[1] Aspects of Syphilitic Nervous Affections. 1874.
[2] Lectures on Nerv. Diseases. [3] Nerv. Dis. and their Diagnosis.
[4] Journal Mental Science, July, 1880, p. 165.

Præcordial pain in 27 males and 32 females = 59 patients, or 50 per cent. of the whole number of cases.

Of the 83 patients with cephalalgia 10 males and 16 females had parietal pain, and 11 males and 8 females suffered from pain in the temples, while 9 males and 7 females suffered from occipital pain. In the remaining 22 cases the headache was felt all over the head.

The peculiarity of the cephalalgia of syphilis, when complicated with epilepsy, is the constancy with which it annoys or agonizes the patient, always being present to some extent, and frequently exacerbated toward night-fall or during the night, generally getting worse until the paroxysm breaks forth, or it may in some instances relent as the storm approaches. Indeed, many syphilographers believe this to be the rule rather than the exception. There is certainly something very typical about these syphilitic headaches which, nevertheless, baffles the descriptions that one would like to give of them. Once seen they can rarely be mistaken for anything else, and even the first view of such a case must impress the careful observer with several salient points. The face, one notices, expresses constant suffering, or at least distress and weariness, and the unrelenting character of the pain seems to crush the patient's vitality of appearance and liveliness with an iron heel. If spoken to, the man who has been resting the head on the hands will either answer slowly and painfully in monosyllables or, gradually raising the face to that of the questioner, give an answer and once more return to his former position. These symptoms are not, of course, pathognomonic, but they are certainly characteristic. The pain, too, is in other ways peculiar, and Charcot[1]

[1] Leçons sur les maladies du système nerveux, tome ii, Deuxieme edition, p. 357. Paris, 1877.

has expressed the opinion that the crossed character of the pain in this disease is of value, as it points to the motor zone. Indeed, he regards this headache as typical of the disease, particularly where it is, as it generally is, bilateral; that is, in both temples or both occipital regions at the same time.

In the place of the headache we may have, as prodromal symptoms, slight loss of memory, unwonted slowness of speech, general lassitude, and especially a lack of willingness to make mental exertion. Somnolence may be excessive, and if any of these symptoms are seen in a person whose history is syphilitic they should be regarded as warnings of an approaching crisis of epilepsy or of some other cerebral disorder. The optic disks should be carefully examined, for in many, but not all, cases evidence of brain disease may be denoted.

This is particularly true of syphilitic epilepsy as contrasted with its other forms.

There is also one symptom which may occur early in syphilitic epilepties, or sometimes only late in the disease, namely, repeated partial, passing palsies, which, while they may be in some cases hysterical, are, in a syphilitic, almost pathognomonic of brain involvement, —a momentary weakness of one arm; a slight drawing of the face to one side, which disappears in a few hours; a temporary dragging of the toe; a partial aphasia which appears and disappears; a squint which to-morrow leaves no trace behind it. A symptom which has been asserted as being frequent in this disease is the common occurrence of nocturnal attacks; indeed, cases have been reported by Charcot[1] and Lagneau[2] where this was the

[1] Leçons des maladies, etc.

[2] Maladies syphilitique du système nerveux. Paris, 1860.

case, but there are similar instances, by the score, in ordinary idiopathic cases.[1]

In syphilitic epilepsy there are often well-marked psychical disturbances with incomplete palsies, which, curiously enough, rarely involve the cranial nerves, as has been particularly noted by Heubner;[2] or there may be an excess of psychical disturbance with a minor epileptic convulsion, and with involvement of the basal cranial nerves. A few writers have claimed that the epilepsy of syphilis can always be distinguished, as it is always unilateral, but this is, of course, incorrect, simply because, as will appear later on, unilateral epilepsy of a non-specific type is by no means rare. Thus it will be evident that syphilitic epilepsy is not sometimes unilateral because it is syphilitic, but because the lesions, under these circumstances, are commonly isolated and in the cortex. The statistics of Echeverria, already quoted a page or two back, in which the details of 118 cases of syphilitic epilepsy are given, also provide us with interesting data concerning these points of which we have just been speaking. Eight cases had fits on the same side as the pain in the head, and 11 had fits on the opposite side, while 7 were generally convulsed. Two males, with supra-orbital neuralgic pain, had fits on the same side, 4 on the opposite side, and 13 had general fits. In 5 cases with occipital pain there were unilateral fits.

Lasègue[3] has strongly insisted that all chronic epileptics show some cranial deformity of a more or less constant and well-marked type, and he has proven, to his satisfaction, that the most common change is prominence of the frontal bone on the right side and depres-

[1] Echeverria's statistics contradict the assertion that syphilitic epilepsy is commonly nocturnal. In 118 cases he only found 7 instances.

[2] Virchow's Archiv, Bd. LXXXIV, p. 269.

[3] Annales Médico-psychologique, Sept., 1877.

sion of the malar bone of the same side, with relative prominence of the left malar bone, resulting in torsion of the face and obliquity of the palatine arch, of which the axis deviates to the right side. Garel[1] has also attempted to show that such changes are present. The following statistics were collected by him:—

	Epileptic.	Non-epileptic.
Frontal eminence prominent,	57 per cent.	51 per cent.
Malar prominence on left side,	53 " "	31 " "
Rotation of face,	49 " "	39 " "
Deformity of palate,	32 " "	23 " "
Flatness of one side of face,	71 " "	51 " "
Orbit level,	35 " "	47 " "

Carrying his studies still further, Garel seems rather to contradict these results, unintentionally, for he found an opportunity to examine the tracings of 255 measurements of heads taken by hatters, and found that the right frontal eminence was most prominent in three-fourths of the cases. As this is, however, a greater number than those found in epilepties, namely 57 per cent., it becomes evident that the conclusions of Laségne and Garel need confirmation by further study, for in the measurements made by hatters very few epilepties are included, and their measurements may be taken as representing the shape of the normal head. Garel found the V-shaped maxilla, so common in idiots, only very rarely in epilepties. Studies still more recently carried out by Bourneville and Sollier [2] have confirmed the research of Leségue.

Jacksonian Epilepsy.—By the term Jacksonian epilepsy we mean an affection which separates itself from true or ordinary idiopathic epilepsy by several peculiarities. By far the most important of the peculiar signs

[1] Lyon Médicale, Jan., 1878.
[2] Le Progrès Médical, September 22, 1888.

is the character of the onset, which always begins, in the typical Jacksonian disease, in some peripheral portion of the body, and most frequently in the muscles of the thumb or hand, so that for the moment the movements of the part are localized, and may remain localized at the point of origin, or immediately diffuse themselves over muscle after muscle until all the arm, leg, or other groups of muscles are involved. It is of the greatest importance, however, that the reader should keep the *aura* of an attack separated in his mind from the *onset*, remembering that the term onset is here used by the writer to designate the beginning of the period following the aura, if there be one. Jacksonian epilepsy may be of almost any severity, as, in rare cases, but one muscle may suffer throughout an entire attack, or in others the entire body may be at last convulsed. There may or may not be loss of consciousness, its presence or absence being dependent upon the seat of the lesion in the brain and the severity of the attack. In those instances where only a few localized muscles are involved consciousness is more commonly preserved than lost.

No better way of bringing forward the disease in a clear manner to the reader's mind than to detail a typical case. A. E., aged 24, one year ago first noticed that several times in the course of a week, which was preceded by great anxiety and exhaustion of mind, he had a peculiar sensation,—tingling, which passed up his left arm, to be lost in the trunk, and at this moment, entirely without his will, his thumb was turned into the palm of his hand in a spasmodic manner, and that afterward the same hand felt weak and weary, as though he had used it very severely all day. These attacks were followed in the next few weeks by several more, but the last one of these was only a little

more severe than the first. Six months ago, after a long walk in the hot sun, he felt the sensation creeping up his arm more rapidly and severely than ever before, and immediately afterward the thumb was once more turned into the palm of the hand, the museles of the hand, wrist, and forearm were rapidly involved in a tonie spasm, which soon relaxed, to be followed by clonic spasm, by which all the muscles were affected, and the museles of the arm and shoulders also beeame involved. After this attack the same feeling of uneasiness came on as after the previous and less severe attacks, except that the sensation of weariness involved the entire limb instead of only the hand, as had been the case before. There was, however, no disturbance of eonsciousness, and only a momentary giddiness at the instant that the aura reached the head. Sinee that time he has had in all six attacks, each one of which has been more severe and more widespread than its predeeessor, and in each of whieh the body became more involved in the movements. In the last two attacks there has been partial loss of consciousness, and they have been followed by the peculiar somnolent condition so frequently seen after ordinary severe epileptie paroxysms. In the last attaek the whole body was equally convulsed before the seizure ended, and if any one had seen the case at this time it would not have been possible to have distinguished it as a case of Jacksonian epilepsy, unless the history already given was known.

This, then, is a ease of Jacksonian epilepsy, which is typical in all its details. The previous history of the man is that six months before his first attack he received a kick on the head, in the parietal region, on the right side, which made him unconscious for some hours.

A very interesting statement has been made by Unverreicht, namely, that whatever the course of involvement is, up or down on the side first affected, it is always ascending on the side last affected.

Petit Mal.—Petit mal differs in no way in its essential characters from epilepsy of a much more highly developed form, but in its minor characteristics it is sufficiently at variance with *haut mal* or *grand mal* to separate it in the minds of clinicians. In its most common form *petit mal* consists of a momentary loss of consciousness, accompanied by pallor, or more rarely flushing of the face. The man who is subject to the disease suddenly stops what he is doing for a moment or two, and then takes up his work or subject as soon as he recovers, and at the point where he ceased, being often unconscious of the break in his conversation or labor. Reynolds[1] has divided this minor form of the affection into two divisions. In the first, he places those who are attacked and have no evident spasm, and in the second group are those who have evident spasm. The seizures are characteristically fugacious, and if any spasm is present it is nearly, if not quite, always of the tonic variety. Sometimes the disorder of motility lies chiefly in an inhibition of an act about to be performed. The fork in a man's hand at a dinner-table may be raised half-way to the mouth, then held in mid-air for a moment, and then as the attack passes away continues on its journey to the mouth; or, a woman playing the piano may suddenly pause with her fingers raised from the keys, miss the time of three or four bars, and then go on exactly where she left off, as if no interruption had occurred.[2] Even lighter manifestations may exist than these. A man may pass by, in conversation, a joking saying of a

[1] *Loc. cit.* [2] For such a case see Georget. Malad. Nerveux, p. 384.

friend unconsciously, or, disregarding the reply given to a question, ask it a second time. To this light grade of petit mal may be added the interesting case, recorded by Hughlings-Jackson, of a man who blew his nose upon a a piece of paper and gave the conductor of an omnibus £2 10s. instead of two-pence half-penny. Such attacks are, however, hardly to be called, strictly speaking, forms of petit mal, and, notwithstanding the classification given it by so eminent a man as Hughlings-Jackson, it seems to us to belong to what is more commonly called temporary mental aberration, of which we see so many instances, in the old, who certainly are not suffering from petit mal. In this case, just mentioned, there was very evidently no dimming of cerebral power, but a perversion of its direction.

A very important matter in obtaining a patient's history in cases of petit mal is to discover the presence or absence of sensations described by the sufferer as "faints," "losses," "times," and "giddiness,"—which symptoms may be all of the attack, or, at any rate, all to the patient who fails to recognize the succeeding unconsciousness.

The writer believes it is true that well-developed auræ are quite as common in petit mal as in other forms of epilepsy, but this has been denied by some writers. Some believe it to be more commonly present, while others think it less common than in the fully-developed disease. Petit mal may also affect chiefly the sensory apparatus, and, in these cases, as in reality in all cases, it has but a slim boundary-line between it and grand mal. Thus, S. Weir Mitchell, the well-known American neurologist, in his work on "Nervous Diseases," has described cases in which the whole paroxysm was sensory. In the most pronounced of Mitchell's cases an

aura beginning at the feet passed up to the head, when it was lost in the sensation of a loud sound, like that of a pistol-shot, followed by a momentary sense of deadly fear. It has been thought that such instances partook largely of a hysteroidal type, but there can be no doubt that they eventually become truly epileptic in some cases. Thus, the writer remembers a case occurring in a lad under his charge in the Children's Dispensary of the University Hospital, who was a ragpicker, and who was always attacked on each day, as he sat on the floor picking rags, by a momentary unconsciousness followed by a sensation of intense emptiness of the stomach. This case eventually developed well-marked epilepsy. A very well known writer and friend of the author of this essay had under his charge a child of eight years who would cry out of a sudden with pain in the stomach, become excessively pallid, run to his mother and be held for a moment, when the whole attack would be over. In some of his spells he had tonic contractions of the feet and neck, but never unconsciousness, the paroxysms being followed by heavy sleep. These cases of gastric aura frequently end in true epilepsy.

A very interesting table of the chief characteristics of petit mal has been compiled by Gowers,[1] and is worthy of insertion here. The symptoms are arranged in the order of frequency. The first of the list occurs in one-third of the cases and the second in a sixth, so that these two are the characteristics of about half the entire number of minor seisures met with:—

1. Sudden momentary unconsciousness, or fainting, or sleepiness, without warning.

2. Giddiness.

[1] Nervous Diseases, p. 690.

3. Jerks or starts of the limbs, trunk, or head.
4. Visual sensation or loss of vision.
5. Mental state; sudden sensory fear.
6. Unilateral peripheral sensation or spasm.
7. Epigastric sensation.
8. Sudden tremor.
9. Sensations in both hands.
10. Pain or other sensation in the head.
11. Choking sensation in the throat.
12. Sudden scream.
13. Olfactory sensation.
14. Cardiac sensation.
15. Sensation in nose or eyeball.
16. Sudden dyspnœa.
17. General "indescribable" sensations.

The urine is very frequently passed in petit mal; more commonly than in grand mal, and more commonly in females than in males. When it is passed, the accident almost invariably is accompanied by loss of consciousness.[1]

Under the next heading of Noeturnal Epilepsy are given the records of a case of the writer's which is, typically, one not only of petit mal and noeturnal epilepsy, but also of the manner in which the minor disease may rapidly become major, even in an adult well along in years.

The after-symptoms of petit mal consist mainly, when they occur, in the patient being dazed or stupid for a few minutes, or by his making random remarks, or automatic and causeless movements, which are, however, purposive in their character. These movements are to be separated from those constituting the true attack,

[1] For a full consideration of the frequency of this and other similar accidents in epilepsy, see interesting paper by Rengade and Reynaud, Gazette Hebdomadaire de Méd. et de Chir. de Paris, Janvier, 1865.

although very eminent neurologists have thought otherwise, among them Esquirol, who called these symptoms "masked epilepsy," or Morel, who named them "epilepsia larvata." The cause of these post-paroxysmal phenomena is, of course, unknown, although several very prominent authorities [1] have believed them to be due to temporary failure of the highest centres to control those below them, which consequently act in an automatic manner.

The relative frequency of petit mal to haut mal has been placed by well-informed writers as 1 to 2.[2]

Nocturnal Epilepsy.—In some cases of epilepsy the seizures for many months may escape notice by reason of their occurring at night, while the patient is asleep. As a rule, they are finally discovered either by an extension of the attacks to waking hours or to the presence of a bed-fellow, who is aroused by the movements or cries of the patient. Very frequently a grown man or woman will, on awaking in the morning, find that they have wet their bed during the night, or, more rarely, passed out the contents of the rectum. They complain on these mornings of a sensation of weariness and intense lassitude, "as if they had been beaten or bruised," and the tongue is often tender and swollen from being caught between the teeth. In some cases it may be difficult to rouse the man or woman sufficiently to make them get out of bed, while in others an insufferable headache is present. As has already been stated, nocturnal epilepsy is almost always followed by diurnal attacks later on. It has been asserted that incontinence of urine only occurs in these cases, but this is incorrect, although I believe it is more common here than elsewhere.

[1] Anstie, Thompson-Dickson, and Hughlings-Jackson.
[2] See Foville and Delasiauve, *loc. cit.*

A very useful and valuable series of conclusions in regard to nocturnal epilepsy has been reached by Echeverria,[1] which is now given:—

1. It is more common in females than in males, and almost always is associated with diurnal vertigo, petit mal or haut mal, when it is not with insanity.

2. Its etiology is essentially encephalic, due to traumatism, heredity, alcoholism, syphilis, or strong emotional causes.

3. Nocturnal incontinence of urine, laceration of the tongue, and petechial eruptions on the face and neck are not constant, but when present are pathognomonic.

4. Sudden explosions of frantic momentary bewilderment in the middle of the night, during sleep, or of insanity on getting up in the morning, are proofs of nocturnal epilepsy.

5. Most sleep-walkers are persons of emotional temperament, and ultimately arrive at epilepsy or insanity.

6. The nocturnal epileptic generally forgets the fit, but remembers the frightful dream that accompanied it.

The following history of petit mal passing into nocturnal epilepsy and finally into the ordinary fully developed diurnal attacks is of interest in connection with what has been said in the last few pages. As the case was not only the writer's patient for something over three years, but also a life-long servant in a near relative's family, the author is confident of the accuracy of the account, and of the early history of the patient. At the time she first really began to suffer from these attacks she was 33 years of age, and well preserved for a woman of her years. Strong and capable of hard work, although she had been for many years only a child's nurse.

[1] Journal Nervous and Mental Diseases, January, 1879.

Early in 1883 she first began to complain of attacks of giddiness, in which she felt light-headed for a moment or two, on rising from bed or the dinner-table, or stooping to pick things from the floor. An inquiry into the state of her health gave negative results, as every function seemed in perfect order; menstruation was normal, as were also the intestinal and digestive organs. Her food was good and plentiful, and her appetite was very fair. Although 33 years of age, she had been a child's nurse for over half that time, and had been away from the city for five months every summer in a healthy watering-place with her employer's family; so that her opportunities for good health were great for one of her rank in life. So far as close-questioning, frequently repeated, could go, there was no specific history, either inherited or acquired; nor was there any evidence of hereditary tendency among her family to nervous complaints. She said she had once heard that a distant cousin was epileptic. Repeated, careful urinary analysis failed absolutely to show any abnormal condition of the kidneys, although the morning urine after a night attack was always thick with phosphates. The genital organs were carefully examined, with negative results, and the undoubted chastity of the woman was confirmed by the finding of fairly well marked and perfect hymen. The administration of iodide of potassium to remove the faintest possibility of syphilis was only productive of iodism, although small doses were given. The use of the bromides held the dizziness in check for some months, so that it got no worse, but did not relieve them, and it was found necessary to increase their amount constantly. She now began to complain that frequently, when sitting with the other servants, perhaps at a meal, she would suddenly become flushed, then pale, and simultaneously

lose consciousness to a slight extent. The voices of her companions seemed afar off; she could hear them speak to her, but could not answer. When the attack passed off, after lasting, perhaps, thirty seconds, she could reply to what they had said, but felt heavy and dazed for some time afterward. These attacks became gradually more severe and more frequent, but never produced any true epileptic seizure.

In the month of January, 1886, the writer was roused at about 1 o'clock in the morning by a messenger asking him to come to the house where the woman was employed, "as they could not wake her." It was the woman's custom to sleep in a room with one child, while a grown sister occupied an adjoining room. This sister had been waked up by strange sounds issuing from the maid's bed, and, finding herself unable to rouse the woman, waked the rest of the household. By the time the writer arrived, however, he found the patient sitting up in bed perfectly conscious and receiving explanations from the family, gathered in the room, as to the cause of the excitement, of which she knew nothing. Nothing further occurred of note until the following June, while away from home, the family being by the sea, when the parents were roused about 3 o'clock one morning by screams of the little girl, who still was accustomed to sleep with the nurse. The child had been wakened by the seizure of the woman, had tried to waken her but failed, and then, becoming alarmed, had called for help. The father broke open the door and, finding himself unable to waken the woman, carried the child to his room. In the morning the woman knew nothing of the attack, but felt tired and heavy. Again another period of six months elapsed, during which time she was without treatment, as the writer was away from home, during which no seizure took

place, as far as is known, when she again roused the household. After this she became thoroughly alarmed, as her tongue was badly bitten, which impressed her greatly as a circumstantial evidence that the nocturnal attacks were as severe as had been stated by the family. No attacks ever occurred where there was relaxation of the sphincters. The attacks of diurnal petit mal were now so frequent and severe that they necessitated her discharge from service, but she remained under the writer's care. Her eye-grounds were most carefully gone over by a distinguished oculist of Philadelphia, who found absolutely no signs of eye or brain disease; indeed, he said "few people have such normal eyes as she has."

Under the active use of the bromides only two attacks at night were suffered from in the next six months, and as the attacks of petit mal were less frequent and severe she once more sought employment. All went well for some two months after this, until on a certain evening the people of the house, having left her as the sole occupant, came home late, could not make themselves heard, broke in the front door, and found the patient under the kitchen-table surrounded by all the evidences of a fully developed epileptic seizure. Other attacks have since occurred in the day-time, notwithstanding the constant use of large amounts of bromides and antifebrin.

It will be seen that this case, which has been given at perhaps too great length, embodies a typical history of petit mal afterward going on to grand mal, and of nocturnal grand mal eventually becoming also diurnal. The case is also of interest as regards the age of the woman, as idiopathic epilepsy rarely occurs after her age, and she certainly had no syphilis.

Epilepsie Apoplectique and Post-Hemiplegic Epilepsy. —These convulsive conditions may be divided into two classes, in one of which the patient suffers from a single convulsion, the result of a cerebral hæmorrhage, and in the other in which the changes produced by the hæmorrhage result in epileptic attacks. When the convulsion occurs at the time of an apoplectic effusion it is generally Jacksonian in character; that is to say, one muscle or group of muscles is involved, or, if not this, the attack is, at most, only unilateral. Further than this, it is always associated with the symptoms of apoplexy as generally seen, for there is inequality of the pupils, drawing of the face to one side, and a consequent hemiplegia which lasts indefinitely. Of the attack itself it may be said that, so far as the movements are concerned, they differ in no way from those of the true epileptic seizure, but it should be remembered that hemiplegia often follows ordinary idiopathic epilepsies; so that the fact that the hemiplegia is permanent, and is not temporary, is more of a sign that the attack is due to hæmorrhage than the actual paralysis is. It should also be remembered that apoplexy often complicates epilepsy, being produced by the convulsion. In a considerable number of cases of epilepsy it will be found that the convulsions succeeded an attack of paralysis, which was sudden in onset and possessed the characteristics of vascular rupture. In some persons the history of this attack is very indistinct, owing to its occurrence in very early life, while in others the paralysis has been so slight or temporary as not to bear any relation in the mind of the patient with the convulsive seizures following, which, in many cases, do not occur for some time after. The palsy may leave not a trace of loss of power behind it, but the convulsions continue, and closely re-

semble the so-called idiopathic form of the disease. The writer also wishes to call attention to the fact that the palsy and convulsions are not always due to hæmorrhage, but to any pathological central change. Heart disease, by causing embolism, may bring it on, and rheumatism, syphilis, and the puerperal state may all produce a softening of the cortex, with an epileptic state following the paralysis. We can very readily divide post-hemiplegic epilepsy into two classes, too, for we find that in about one-half the cases the convulsion occurs along with the paralysis, and then follows at intervals, while in the other half the paralysis is not followed by convulsive seizures for weeks, months, or years.

Post-hemiplegic epilepsy may occur at any age, but there can be no doubt that it far more commonly occurs in infants than in adults. In at least two-thirds of the cases the onset is before 5 years of age, and in nearly one-half it is during the first two years of life. Very curious results are reached if the statistics of the affection are analyzed,—results which are quite unexplainable unless by hypothesis. Indeed, they tend to overturn many of our preconceived ideas. Thus, it will be found that in the cases which date from infancy, females are twice as numerous as males, but in cases after 5 years of age there is no difference between the frequency in the two sexes. One of the theories of these infantile cases has been that they were produced by the use of instruments during labor, and repeated post-mortem examinations have confirmed the possibility of this occurrence. On the other hand, every obstetrician knows that the birth of a boy generally means a more difficult labor than that of a girl, owing to the greater size of the head in the male child. *A priori* reasoning would seem to show, therefore, that the heads of male

children would, accordingly, have the instruments applied most frequently, and consequently that infantile cerebral trouble would be the result more commonly in males than in females; but, as has been said, this conclusion is contradicted by the facts. Another symptom of great interest is that the paralysis, in the infantile cases, is more frequently on the left side than the right, but after the fifth year it is equally common on both sides.

The writer has already spoken of the fact that the convulsions may occur along with the first attack of paralysis and continue, or that an interval may occur between the attack and the subsequent paroxysm. The chronic recurring fits date from the onset in about one-third of the cases, and it is not uncommon for the paralysis to occur in infancy and the epilepsy to begin at puberty. It would seem that cells injured in early life laid undisturbed till the increased demands of maturity called them out into diseased action. This prolonged interval occurring so commonly in children separates them from adults in this disease, for in the latter class it is very rare for the epilepsy to be put off for more than one year.

A distinct aura is present in about five-sixths of the cases, and is consequently far more frequent than in the ordinary idiopathic disorder. When speaking of the etiology of this affection it will be clear to the reader that these conditions are virtually forms of Jacksonian epilepsy so called; at least, so far as the causative lesions are concerned.

The frequency with which post-hemiplegic epilepsy comes on in the hemiplegia of childhood has been very recently studied, and the conclusion reached that its occurrence is quite common.[1] Thus, in Osler's cases,

[1] Osler, Medical News, July, 1888.

20 children out of 97 suffered from it. In the 80 cases collected by Gaudard[1] 11 children had hemi-epilepsy, and 66 children out of 160 cases collected by Wallenburg[2] were epileptic after hemiplegia. In another series of cases collected by Osler 15 children out of 23 were thus affected.

Night Terrors.—Night terrors may be divided into two divisions, in the first of which no further nervous changes occur, and in the second of which true epilepsy ultimately develops. The writer is well aware that the profession, at least in the majority, generally look upon this condition as entirely separate, and apart from partaking of an epileptiform character; but he is equally thoroughly convinced that frequently night terrors may be but an evidence of nocturnal petit mal. He has seen too great a number of cases which followed out to the letter all of the course usually followed by the minor attacks to doubt this for a moment, and he has already pointed out that in nocturnal epilepsy the patient usually forgets the seizure, but remembers the frightful dream which preceded it, showing that a night terror usually occurs even in adults and with completely developed epilepsy. There are, of course, certain children who frequently have nightmare from indigestion or a fright experienced during the day, but these are only occasionally affected, and comparatively mildly so as regards the concomitant mental disturbance.

When a child is brought to the physician with true night terrors or nocturnal petit mal, we generally find the following history: The patient goes to bed apparently perfectly well and as bright as usual, sleeps soundly

[1] Contribution l'étude de Hémiplégie Cérébrale Infantile. Geneve, 1881.

[2] Ein Beitrag zur Lehre von den Cerebralen Kinderlähmungen, Jahrbuch für Kinderheilkunde, 1886.

until the night has progressed to some extent, and then rises up in bed and utters a shriek of the most heart-rending character, which may or may not arouse the child to consciousness. The eyes are wide open and staring, and show by their widely dilated pupils the severity and intensity of the alarm from which the little one is suffering. The face is generally pale and tearless. The parents as they come to the child are clutched and drawn down to the bed by the frightened little one, who trembles, cries, and refuses to be comforted, often crying out that it sees some fearful beast, or begs that some distorted image of the imagination be taken away. A horrible, indefinable dread seems to blot out, as fast as spoken, all the quieting, endearing terms of the parents, and even the casual bystander looks about the room for the fearful object to which the child points in so realistic a manner. Slowly, amid choking sobs, the child becomes pacified, and passes off into sleep by slow degrees, interrupted by frequent starts or sighs of a more pronounced character than the others. The restless slumber now deepens, and may last until morning, or be followed by a repetition of the attack in a few hours. The final history of all such cases ends in epilepsy proper, preceded by lack of vivacity or momentary clouding of the intellect during the day as the disease extends from the night to the day-time.

Cardiac Epilepsy.—The possibility of epileptiform convulsions coming on, due to lesions of the heart, has been recognized for a number of years, and interesting contributions have been made on the subject by such writers as Stokes, Thornton, and Blondeau, although the literature of the subject has not by any means been confined to the pen of these writers.

It will be remembered that the symptoms of cardiac

epilepsy are chiefly characterized by a remarkable slowing of the pulse, so that the pulse-rate has been known to drop from the normal to not more than five beats a minute, and in a case reported by Thornton[1] it was proved by the stethoscope that in the first stage of the attack the heart ceased to beat for many seconds. In some cases the heart's beat can be heard over the apex, but the patient becomes pulseless at the wrist. The respiratory movements are generally at first quickened and then become labored, and, perhaps, stertorous. The temperature of the body falls very decidedly, not only during but before the attack, so that the complaint of feeling cold may be the only warning given of an approaching seizure, and this sensation of extreme cold occurring previous to an epileptic convulsion is, in the opinion of several writers, almost pathognomonic of cardiac epilepsy. The color of the face resembles the coloring of an ordinary idiopathic attack, being at the first pale, then red and cyanotic. The convulsive movements do not generally assert themselves during the period of unconsciousness, but they may do so, and biting of the tongue has been reported. In some instances the heart has been found after death in a state of fatty degeneration, and this, of course, points to a lack of sufficient blood-supply as being the cause of the attack. That the disease is not in every case due to organic failure of the heart is proved by the fact that Charcot has seen such cases occur in which, after death, no cardiac lesions were discoverable.

The question as to whether the nerve-storm originates in the brain-centres or is only secondarily produced by cardiac failure is one which is solved with great difficulty; since it is perfectly possible to imagine

[1] Trans. Clin. Soc. London, vol. vii, p 95.

that disordered nervous centres might produce not only an epileptic seizure, but also exert an influence over the heart, as, for example, by sending a powerful impulse along the pneumogastric nerves. Every physiologist knows, too, that cutting off the supply of blood from the brain by the partial ligation of the principal blood-vessels in the neck will produce violent epileptiform convulsions, and it has also been proved that the convulsions arising in poisonings from the cardiac depressants, which are epileptiform in character, are due to this cause, namely, starvation of the nervous centres, whereby their functions are perverted or set aside. The true cause of the epilepsies, supposably due to cardiac failure, cannot be considered as decided in every case, but it would seem probable that such cases may arise from both the causes mentioned.

The theory of the production of attacks owing to deficient blood-supply has been greatly strengthened by the recent reports of several observers, notably the very recent one of Lemoine,[1] in which he reports 5 cases of cardiac epilepsy in which valvular disease of the heart existed, and where great amelioration of the symptoms or recovery occurred upon the use either singly or together of such cardiac stimulants as digitalis and caffeine.

While the cardiac muscle was under the influence of these drugs the epileptic seizures became much less frequent, and in some cases entirely disappeared, the only symptoms remaining being vertigo on certain movements, which, however, decreased as the condition of the heart improved. Such evidence as this is of considerable weight, as it strengthens very materially the belief that in many instances the convulsion is due to the cardiac failure.

[1] Rev. de Médecine, May 10, 1887.

In a case reported by Lebrun[1] the symptoms resembled those of cardiac epilepsy somewhat in that there was a slowing of the pulse from 70 to 32 beats, with clonic spasm and syncope. After the first attack the pulse never rose above 40 beats, and death followed in a short time. There was no autopsy. Before the attacks there was vertigo, diplopia, epigastric distress, and yellow vision.

Epilepsia Procursiva.—*Epilepsia procursiva* of the older writers has within the past eighteen months been studied most carefully by that accurate observer Bourneville.[2] The disease is one characterized by attacks which consist in a straight or circling run of a variable distance, which is rarely followed by a fall or coma, but by facial congestion. Bourneville classes these cases into those in whom the run is uncomplicated with other forms of seizure, those with a progressive aura, and those in whom there is epilepsy followed by running. He also has studied 25 cases of procursive vertigo. The essay of M. Bourneville is so exceedingly exhaustive that the writer can only refer the reader to the original for further information, but the following cases occurring in his own practice, one of which is under his care at the present time, are of great interest in this connection.

Jesse ——, aged 14, is a strong and apparently healthy boy, of healthy parents. He is a remarkably well-built and manly-looking fellow, of more than the usual height and weight, and well proportioned. His complexion and color is good, and he is notorious at his school for his brightness and acuteness in learning and understanding his lessons. He is not one of those typically "bright

[1] Bull. de l'Acad. Roy. de Méd. de Belgique, 1887.

[2] Archiv de Neurologie.

boys," who look as if highly strung, but is perfectly normal so far as his appearance is concerned.

He was entirely devoid of any morbid condition until just one year ago, when the first fit took place. The history of his life gives no record which can be in any way considered as a cause of his present trouble, save that when he was about 6 years of age he was struck on the back of his head by another and much larger boy, who had a heavy club in his hand. No unconsciousness was produced, but blood flowed freely from the wound, the scar of which is still to be seen about 2 centimetres downward and to the right of the occipital protuberance. No secondary effects ensued from this stroke, but exactly two years ago he was once more struck while at play on the head, within 2 centimetres of the former scar. The blow felled him to the ground and produced unconsciousness lasting for some minutes. One year ago, almost on the anniversary of this last blow, he was down in the cellar of his father's house, when he suddenly rushed upstairs and sat on the kitchen-table, muttering and moving his legs, and apparently unconscious of his surroundings. There was no coma nor headache after this attack, and he seemed perfectly well. The other attacks have since occurred, either once or twice a week, or even less frequently at certain times.

When they occur in the day-time they invariably consist in a sudden run across the room, ending in a fall to the floor, but there are no spasms when he falls. One week ago he was sent to a store for some nails. He entered, asked for the nails, and then suddenly darted out into the street, throwing his money away as he ran. The distance traversed before he was caught was about 100 metres. The author of this essay had up to this time never seen him in an attack; but once, on attempt-

ing to take him before a class of students for a lecture, he immediately had a paroxysm, precipitated by fright.

The head was drawn to the left slightly, the right shoulder thrown forward and upward, and he assumed the position of one about to start on a foot-race. He was instantly seized by the writer, and used the utmost effort to escape, but the efforts were ineffectual, chiefly because they were lacking in intelligence. The movements of the legs, although he was held, were running movements, finally deteriorating into struggles such as those of an angry child trying to break away from a parent. The entire attack was accompanied by muttering, and at the beginning of it the saliva poured from the mouth.

The aura in this case is distinctly visual, and consists in total blindness. Indeed, at the first part of the attack he gropes blindly about for something to clutch. There is no incontinence of urine and fæces. The time of return to consciousness, or, rather, to intellection, is but momentary, and does not last more than sixty seconds. The mental clouding after the attack is so slight and transient as to be scarcely noticeable.

He has had several nocturnal attacks, consisting in turning and twisting in the bed, with muttering, but no running movements.

The second case was as follows: A girl aged 8 years, the child of healthy parents, was brought to the writer by her mother, who stated that the child would occasionally suddenly give a scream, and then dart out of the house into the crowded street and run one or two squares, when she would return to consciousness dazed and unable to find her way home, rather because of her surprise at her new surroundings than an absolute lack of intelligence. At the time spoken of the attacks were

so frequent that the mother could not perform her household duties because of the constant watching necessary. In other words, the attacks occurred several times a day. If held so that escape was impossible the girl struggled to get away, but there were no fits of the ordinary character at any time. There was never any coma after the attacks; no incontinence of urine or fæces. There was no history of any cause of the disease, so far as could be discovered. The mother was told to bring the child back in one week, but failed to do so, and all trace was lost of the case.

Epilepsy Nutans.—*Epilepsy nutans* is a limited variety of the disease, and has a sudden onset while the child is at play, or the adult is at work. It consists simply in a slight drawing of the head to one side for a moment, followed by a nodding movement, repeated several times, after which the child immediately resumes its play. Like several other forms of epilepsy, its character indicates its localized origin in the cortex.

Epilepsy Loquax.—*Epilepsy loquax*, or muttering epilepsy, has been recorded by Cheadle,[1] an English writer, and affects the speech-centres altogether, all the rest of the cerebrum escaping. In the 4 cases seen by Cheadle there was a sudden attack, during which the patient simply repeated one word again and again until the paroxysm passed away.

Epileptiform Migraine.—An irregular and very rare disorder is that known as *epileptiform migraine*, several cases of which have been reported. The writer is, of course, aware that migraine and epilepsy are closely associated, but the cases here spoken of are somewhat different. In these cases there is flexion of the fingers of both hands and numbness of the feet, with violent

[1] Brit. Med. Journal, May 1, 1875.

pain in the head of a hemicranial character. The tongue feels too large for the mouth, and speech is difficult. When writing is attempted (for consciousness is preserved), although words are ready to flow, only meaningless scrawls result. The eye-sight grows dim and the pain in the temples increases in violence, these symptoms being followed by vomiting and deep sleep, from which the patient awakes well. As the attacks go on consciousness becomes slightly dimmed, but is never lost, until the disease is very far advanced.[1]

Toxæmic Epilepsies.—Such convulsive disorders may arise owing to the presence of a very large number of toxic substances, of which the writer shall only here consider a few, as most of them are spoken of most thoroughly in the sections on etiology and pathology. The convulsions of fever differ from the true epileptic attack very slightly indeed. It is only by the history of the patient and by waiting for developments that we can determine which is which, for as soon as the eruption or high temperature of an exanthem occurs the character of the attack is evident.

Alcoholic epilepsy consists of two distinct varieties produced by overindulgence in intoxicating drinks. In one of these the convulsions are symptomatic of acute poisoning, and come on during a drunken orgy, or immediately after a single large draught of liquor. In the second variety the convulsion does not originate while there is alcohol in the blood, but in the intervals between the attacks of delirium tremens resulting from chronic excessive alcoholic indulgence. Under these circumstances the paroxysms are generally accompanied by hallucinations or by dementia or imbecility. In the alcoholic convulsion the symptoms may closely resemble

[1] Such a case is reported by Allbutt, in "Brain," 1883-84, p. 246.

those of true epilepsy, and not rarely the attack is ushered in by headache, gastric embarrassment, disorders of vision, and excessive tremors, or some similar prodrome which may be looked upon as partaking of the nature of an aura. As a general rule these alcoholic convulsions occur in paroxysms,—two, three, four, or more, one after the other, at intervals of a few minutes. Not only may grand mal be closely simulated by alcoholic epilepsy, but simple vertigo or true petit mal may exist, either alone or associated with major convulsions. Alcoholic epilepsy is often associated with hallucinations, especially of terror, and not rarely is followed for days by a certain degree of mental disturbance. Rather curiously these cerebral disturbances result rather in suicidal than homicidal tendencies, which is just the reverse of the insanity following simple epilepsy. It is very important that the reader remember that alcoholism in producing epilepsy very frequently produces a permanent nervous disorder which the withdrawal of the poison will not remove.

The symptoms of uræmic convulsions will be spoken of further when studying differential diagnosis in connection with epilepsy.

Convulsions Produced by Drugs.—As some cases of sudden epileptiform convulsions are apt to result in an official investigation as to their cause, and, as the character of the treatment of the case before death may influence the question of life and death for the accused very greatly, it is well for the physician to bear in mind that certain drugs when taken in poisonous doses produce very violent epileptic convulsions. This is particularly true of the so-called cardiac sedatives, or depressants, such as aconite, veratrum viride, sabadilla, hydrocyanic acid, and one or two similar substances.

Attention will be called to the manner in which these drugs reach this result further on. Suffice it to say that experimental researches have proved that they act by disordering the cerebral circulation.

The symptoms of epilepsy due to chronic poisoning by lead are chiefly as follow: The man, apparently in his usual health, or who has had for a few days a feeling of weight in the head, or headache, is suddenly seized with most violent convulsions, which are often fatal, and which during their presence resemble ordinary epilepsy so closely as not to be separated from it. They end in coma, and are separated from each other by intervals of nervousness and disquiet. In some cases, one convulsion follows the other so rapidly that death ensues from exhaustion, but in much more rare instances the attacks may resemble Jacksonian epilepsy very closely, and there may be no loss of consciousness. If such a condition occur, it is almost sure to be followed by a more violent fit. The attacks are not preceded by any aura whatever, but previous to the headache, already mentioned, the patient may have had amaurosis, and ophthalmoscopic examination of the eyes may show choked disk and neuritis of the optic nerve.[1] As a general rule such cases are fatal, but they may recover under careful treatment.

Malarial epilepsy is an uncommon disorder, even in countries and regions which are notoriously malarial, but it does undoubtedly occur, particularly in the southern part of the United States and in Brazil. The only cases which the writer can find recorded are by American or English observers, namely, Jacobi,[2] Payne,[3]

[1] Norris, Amer. Syst. Pract. Med., vol. iv.
[2] Hospital Gazette, New York, v, 41–43.
[3] Indian Ann. Med. Sci., Calcutta, 1860–61, vii, 597.

and Hamilton.[1] The latter gives but a passing glance at the subject, and the articles of the others the author has not been able to obtain; so that he knows them solely by reputation. In Hamilton's case, a young man who had lived for many years in an exceedingly malarious region had more or less periodic epileptic attacks, attended by great preliminary rise of temperature and intense congestion of the face and head. He was unusually somnolent, and in the intervals frequently suffered from facial neuralgia. Change of the place of habitation and the use of quinine removed the disease entirely.

Spinal epilepsy, as it has been termed by some writers, really never occurs, but under this heading the writer intends to place those interesting cases first recorded by Charcot,[2] in which periodic epileptic attacks come on in persons suffering from spinal lesions particularly due to compression. Contrary to what might have been expected from the well-known effects of section of the spinal cord on one side in certain animals, epilepsy in man from spinal lesions appears to be very rare, but it does occur. Charcot has seen 10 such cases, 5 of which were due to disease of the cervical part of the spinal cord, produced by compression. As this well-known neurologist has said, these convulsions are really quite separate from those produced by Brown-Séquard in guinea-pigs, for in the latter the movements are limited to the part below the injury, while in the cases recorded by Charcot and others the whole body is affected.[3] Herewith is appended a list of several such cases for the

[1] Amer. Syst. Pract. Med., vol. v.

[2] Leçons sur les maladies du système nerveux, series ii. Paris, 1873–77.

[3] It is to be remembered that this assertion is not borne out entirely by facts. I have seen guinea-pigs with spinal hemisection suffer from very wide-spread epileptic convulsions.

use of those interested, particular attention being paid to the very remarkable case reported by Duménil, of Rouen.[1] In the following references the spinal lesions were in the lumbar or dorsal cord :—

Leudet, Arch. de Méd, tome i, p. 266. 1863.

Ollivier, d'Angers' 3d edition, tome ii, p. 319. 1837.

Rilliet and Barthez, tome iii, p. 589. 1859.

Michaud, Sur la Meningitis et la Myélite, p. 50. Paris, 1871.

Brown-Séquard, Recherche Epilepsie, p. 11.

Westphal, Archiv f. Psychiatrie, p. 84. 1868.

Etiology of Epilepsy.—As has been said elsewhere in this book, the question as to what is the cause of epilepsy has been asked for centuries without receiving any answer of a satisfactory character. Isolated cases or groups of cases have of course been explained, as, for example, to a certain extent, the traumatic and reflex disorders; but the cause of idiopathic epilepsy still remains to be discovered, either in its original character or ultimate results. We are, therefore, when dealing with this branch of the subject, forced to use great care in keeping our judgment unembarrassed in order to exclude all unimportant and extraneous points, which have been so apt to retard advancement in the past.

It is a characteristic of the human brain that whenever it fails to explain a condition it begins to "beat around the bush" and to invent some hypothesis which has no basis at all, or by means of a species of reasoning of the *post hoc procter hoc* character it attempts to fathom unfathomable depths. Scarcely a day passes that the physician is not tempted to carry out such a line of argument, often because it seems most natural, and, while in some instances the result may be of a

[1] Quoted by Charcot, *loc. cit.*

true nature, in epilepsy it is very frequently at variance with facts. Of course such faults are present in the discussion of many diseases, but the very insidiousness of epilepsy not only aids in driving the physician to hypotheses, but also renders his argument likely to fall to the ground. Both patient and physician are often persuaded to accept an opinion which is surrounded by circumstantial evidence, but which otherwise has no claim upon their consideration.

A point, whose importance cannot be estimated, is the distinction to be made between indirect and direct etiological factors, and this is emphasized when the indirect factors may at times play very important rôles. This may be made more clear if an example be given. It is not at all uncommon to find cases of epilepsy in which the patient insists most strenuously that he or she were in perfect health until a certain period, when they received a severe fright or were struck violently upon the head. Almost invariably such persons are persuaded absolutely that this incident is the entire cause of all their suffering, and even infuse the physician with some of their belief that such is the case.

Now, it is evident that this is true sometimes, and that in many others it is entirely separate from the disease; it may be the direct or indirect cause, or it may have no connection with the disease save that of coincidence. The manner in which these accidents may become the direct cause is widely recognized by every writer, and it cannot be doubted that in a certain number of cases organic changes in the nervous system may be thus produced, for we have not only an abundance of incontrovertible evidence of a clinical character, but also of a pathological nature. That the change does actually occur in some instances seems proved by the fact that,

as a general rule, the first paroxysm does not follow the fright for some space of time, and the longer this is deferred the more distinctly are the attacks epileptic. Whenever a fit comes on immediately it is probably hysterical. We see, therefore, that fright or a blow may be the cause of an attack in one case and not in another. Naturally if two cases have a history of cerebral injury, and both are epileptic, one is apt to think that the same cause is at work in both instances, but in reality no proof exists that such is the case. In one individual elevation of a depressed fragment of bone may result in a cure; in the other, even though a depression exists and is removed, no benefit may be reached, because, in the period between the reception of the injury and the first fit, the man has contracted syphilis, which has produced epilepsy, under the cloak of traumatism, and which is only relieved by mercury and the iodides, or he has a constitutional inherited tendency to epilepsy, aside from any extraneous causes. The writer has seen two boys both with adherent and inflamed prepuces, and both with epilepsy: in one circumcision produced a cure; in the other this operation was absolutely negative in its results, and it was ultimately found that this case inherited strong epileptic tendencies.

What the writer has said has not been with the object of rendering any opinion hazardous, but with the idea constantly before him that everything must be closely looked into before it is put down as a causative factor. Keeping this in mind my readers will have no trouble in reaching fairly definite conclusions in every case.

Returning to the question as to whether fright may really produce organic epilepsy, and the writer has already answered this partly in the affirmative, we find that it

may do so. Thus, in one case recorded by Wood,[1] a child was, at the age of 2 years, taken some distance in a railway train, which frightened it very much, so that it kicked and screamed and finally became convulsed before the destination was reached. Nothing further occurred save that the child showed evidences of arrested development and afterward became epileptic. As it grew older it failed to talk, and died in childhood. At the post-mortem examination the region of the brain where the speech-centre should have been was found scooped out as if by a knife. In other words, that portion of the gray matter was atrophied as a result of a fright. There was no history of any character whatever pointing to any other cause for the nervous changes, and the child had been previously a picture of health.

There can be no doubt of the influence of fright in some instances, therefore; but even here we do not know but that there had been some subtle change going on which only needed the mental disturbance to ignite the morbid process into flame. If this is true we also find that the severity of the provoking cause does not of necessity bear any relation to the likelihood of producing the disease, provided the unrest of the diseased centres is just on the verge of an eruption. A mere peristaltic wave passing suddenly along the intestine may be the necessary spark, and, while this for the moment seems unlikely, there is good evidence that it is not impossible. Such a cause may bring on a paroxysm in strychnine poisoning with the greatest ease, simply owing to the fact that the reflex centres are in an excited state.

Passing on to the consideration of the more direct

[1] Nervous Diseases. 1886.

causes of the disease we find that several exist, although so far we have only proof of one or two. Indeed, in many instances of idiopathic epilepsy no cause can be determined, and we are forced to confess, every now and then, that we are completely baffled and compelled to make a group of cases known as the apparently causeless.

Heredity.—Among the causes which may be said to be pregnant with epilepsy is heredity, and all observers are so in accord with this statement that it seems almost useless to offer evidence of the truth of the assertion. However, the following points, bearing this statement out, are of great interest, and properly belong in such an essay as this: Herpin [1] found that in 68 cases seen by him 10 were descended from epileptic parents, and Delasiauve [2] found 33 hereditary epilepties in 300 cases of the disease. In 171 epileptics seen by Hammond,[3] 21 had epileptic fathers, mothers, grandparents, uncles, aunts, brothers, sisters, and 24 had relatives insane, hysterical, cataleptic, or suffering from hemierania. Echeverria,[4] in his collection of 306 epilepties, found that 80 had a hereditary tendency, or 26 per cent.; and Hamilton,[5] another American neurologist, goes so far as to assert that 50 per cent. of all the 980 cases seen by him were hereditary. In the statistics collected by Martin,[6] Boucher, and Cazanveilh,[7] it was found that 19 epileptic parents begat 78 children, of whom 55 died very young and generally in fits. Of the 23 remaining, 15 only were healthy, and they were all very young; and this is prob-

[1] Pronostic et du Traitement Curatif de l'Epilepsie, p. 325. Paris, 1852.

[2] Traité de l'Epilepsie, 1854.

[3] Dis. of Nervous System.

[4] On Epilepsy: Anatomo-Pathological and Clinical Notes. New York, 1870

[5] System of Medicine, Pepper, vol. v, p. 468.

[6] Annales Méd. Psycholog., 1878 Novembre to 1879 Septembre.

[7] De l'Epilepsie Considérée dans les Rapports avec Alienation Mentale.

ably the reason why Esquirol and others have failed to find, exeept rarely, a hereditary tendeney in the adults examined by them.

By far the largest number of cases eollected by any one writer, so far as the author is aware, are those of Gowers,[1] who analyzed no less than 1450 eases of epilepsy, finding that an inherited tendeney was indicated by the presenee of insanity or epilepsy in aneestors or eollateral relations in rather more than one-third of the eases (35 per cent.), and rather less frequently in males than in females, for there was this history in 33 per cent. of the males and 37 per eent. of the females. There was a family history of epilepsy in two-thirds of the inherited eases, of insanity in one-third, and both disorders in one-tenth of the eases. In the 56 eases reeorded by Sieveking[2] heredity was the cause in 11. Reynolds,[3] in his eolleetion of eases, found the proportion to be 31 per cent., while Nothnagel,[4] who seems to write rather from memory than from actual figures, agrees with him. Hasse,[5] another German writer, has eollected 1000 cases, and has found heredity the eause in no less nnmbers than the others. If we take the average result of the eonelusions reaehed by the elinieians just named, who give exaet figures, we find that we have to deal with 4300 cases of epilepsy, of whom a little over 26 per eent. were due to heredity. It is evident that the transmission from parent to ehild is most frequent, and, to impress this faet on my readers, let me detail a ease reported by Gray.[6] It is that of a married woman aged 40 years, who began

[1] Epilepsy and Other Chronic Convulsive Disorders. London, 1881.
[2] Epilepsy. London.
[3] Treatise on Epilepsy. London.
[4] Ziemssen's Encyclopedia, art. Epilepsie.
[5] Krankheiten des Nervenapparates, Virchow's Handbuch f. Special Pathologie und Therapie.
[6] Journal of Mental Science.

at the age of 12 years to have epileptic attaeks about every three weeks, in whieh she saw demons with the right eye. She had, after marriage, nine ehildren, who all died as follows :–

First, a girl, died on the fourth day of life in convulsions.

Seeond, a boy, died at 11 months in eonvulsions.

Third, a girl, died at 15 months in eonvulsions.

Fourth, a boy, died soon after birth in eonvulsions.

Fifth, a girl, died soon after birth in eonvulsions.

Sixth, a boy, died at 5 months of age in eonvulsions, having been eonvulsive all his life.

Seventh, a girl, died after birth in eonvulsions.

Eighth, a boy, died after birth in eonvulsions.

Ninth, a boy, died after birth in coma.

Close-questioning failed to elicit any other neurotie family history than that given, and there was no speeific history.

While the writer does not desire to deny in any way that heredity is a remarkably eommon factor in the production of epilepsy, it is his duty to point out that all the writers quoted are not on the same footing as regards what may be included in hereditary influenees in the parents. Hamilton, who, it will be remembered, found 50 per cent. of 980 cases to be hereditary, placed every case in whieh there was a family history of insanity, phthisis, epilepsy, eerebral apoplexy, tumor of the brain, or some lesser neurosis, in his list of hereditary cases. This is manifestly wrong, since eerebral apoplexy or phthisis, in reality, has very little to do, as a general rule, with epilepsy in any manner whatever.

That the inclusion of apoplexy in this list is entirely erroneous is proved by the eareful investigations of Herpin, who found that the frequeney of apoplexy

among the relations of epilepties was positively smaller than in the population at large.

However, there can be no doubt that other diseases than epilepsy in the parent may produce hereditary epilepsy. Nearly every writer states this fact, and some, like Hamilton, carry it absurdly far, as, for example, Nothnagel.

The very existence of other forms of nervous disease than those which are convulsive in character also has an influence on the offspring, but not to such an extent as those with which spasm is associated; insanity is probably the chief one of these. A neurosis so mild in the parent as to escape notice may blossom out in the offspring into epilepsy, and hysteria, hypochondriasis, and catalepsy frequently stamp epilepsy indelibly upon the children. Migraine is a very common history in some cases; that is, where the migraine is really migraine, and not simply severe headache confined to one side of the face. Nothnagel[1] gives a very interesting case, illustrating this, in which a woman suffering from this disease had an epileptic son and a hysterical daughter. He insists that there was absolutely no other predisposing cause present; but one can hardly agree with him when he speaks of simple neuralgic pains in the parents as a hereditary excitant of epilepsy, or of mere nervousness as a parental influence, except in a very indirect manner.[2]

Just here the question may be asked, What do we mean by heredity? It is, when correctly used, a term signifying the transmission of a particular peculiarity, or peculiarities, from parents to their children. On the other hand, inherited predisposing factors are more in-

[1] *Loc. cit.*

[2] Wagner and Strümpel have both denied that any relationship exists between the two.

directly at work, and may bear no relation to the peculiarity in the child except in a general way. It is to the latter class that all the distant neuroses of these writers should be assigned, for they only prepare the system of the child for epilepsy. High-strung, nervous races should by this argument have a larger proportion of epilepties than those of a more phlegmatic character, and every one knows that this is untrue by the statistics which have been published of the prevalence of epilepsy in various nations from time to time, and to which the writer will refer more fully again.

Within the last two years, however, Marie[1] has written an article strongly combating the idea that heredity is a strong or frequent cause of epilepsy. He believes that the tendency is always post-natal, but the writer does not think that his deductions are warranted by his arguments, and they can hardly overturn the experience of a very large body of observers.

Some difference of opinion exists as to whether the mother or the father transmits the epileptic condition to the offspring the most frequently. Gowers and Hamilton both state that the inheritance is more frequently from the mother than the father; whereas, Nothnagel asserts that there is no such difference, believing that the condition is equally transmitted by both sexes. The first of the English writers makes rather ambiguous statements, which, owing to his eminence as an authority, are herewith quoted, hoping that the reader will be able to fathom his meaning. He says: "Where there is an inherited tendency, the females of a family are rather more likely to suffer than the males. This is due to two circumstances; the inheritance is rather more frequently from the mother's side than the father's, and more

[1] Le Progrès Médical, 1887.

females suffer when the heredity is maternal, more males when it is paternal." He then goes on to say that "the inheritance is less frequently from the mother than from the father; but this is due to the fact that insanity is much less common in the mother than in the father; epilepsy is equally common in both."

It will be seen that this directly contradicts that which has been said by the same writer but a few lines back.

Sex.—Another point, in connection with the question as to which sex suffers from epilepsy most frequently, is still undecided, although the statements of each body of statisticians include large numbers of cases. Reynolds, in his collection of cases, found no difference in the sexes, as did also Nothnagel and Herpin.[1] On the other hand, Gowers, in his collection of 1450 cases, found that there were 114 females to every 100 males.

Rather absurd deductions as to the relative frequency of epilepsy among the sexes have been drawn by no less noted observers than Esquirol and Moreau, who examined cases at Bicêtre and the Salpêtrière. At the first there were 311 epileptic males, and at the second 723 epileptic females. From these facts they deduce the conclusion that epilepsy is more frequent in females than in males, which is incorrect in fact, and is reached by erroneous methods of calculation, as they do not seem to have reckoned the number in each asylum in respect to their proportion to the population. The only other figures which the writer has been able to find in regard to this point have been collected by Englishmen, and in each instance combat the views of Gowers, Esquirol, and Moreau, placing males as the most common sufferers.

[1] It is just to say that while Herpin uses these words his figures really disagree with those of Gowers, for he found the proportion to be 110 males to 96 females.

Boyd[1] examined 1300 cases, and found among them 145 epileptics, of whom one-third more were males than females.

Althaus[2] has also attempted to decide this question, and has examined an enormous amount of statistics to obtain his results. He divides the cases into periods of five years, as follows, and the results are, I think, conclusive, notwithstanding Gowers's contradiction :—

Periods of Five Years.	Males.	Per Cent.	Females.	Per Cent.
1847 to 1851, . . .	4,479	1.86	4,188	1.74
1852 to 1856, . . .	5,441	2.10	3,998	1.55
1857 to 1861, . . .	5,972	2.41	5,717	2.10
1862 to 1866, . . .	6,585	2.21	5,774	1.96
1867 to 1871, . . .	6,483	2.10	5,805	1.87
	28,960	2.13	25,482	1.84

Phthisis.—It has been claimed by those who ought to know that a family history of phthisis is a cause of epilepsy. Among such believers may be mentioned chiefly English and American writers, for the theory does not seem to be generally received on the Continental side of the channel.[3] Hamilton, the American, in the 980 cases collected by him, found 230 were phthisically inclined, owing to parentage; but he confesses that, as most of his cases occurred among a very tubercular class, his results may be too sweeping for general application

Even if these results are true it does not prove that such a history is pregnant with epilepsy, but only that he practiced among a depraved, half-starved class in a great city in which consumption and epilepsy might well walk hand in hand. As against these views we have arrayed most of the French writers, as well as the German and one or two English authors. Nothnagel and

[1] Asylum Journal of Mental Science, 1875, p. 282.
[2] Diseases of Nervous System, p. 222.
[3] Echeverria, Anstie, Bastian, Savage, and Hamilton.

Gowers both insist that phthisis has no influence on the disease, and they point out, as the writer has just done, that it is only the association of the two diseases that binds them together.

Alcoholic Influences.—Another form of hereditary taint in this disease is the ALCOHOLIC habit in the parents, and here we are brought to another cause of epilepsy, which may be placed under the class of predisposing causes or of heredity. It is evident, too, that in this condition epileptic children may result from the insanity of rum as well as the ordinary type of insanity and mental disorder. Unfortunately, the relation of epilepsy to alcoholism does not end here, for the drug may not only produce epilepsy by inheritance, but directly in the drinker himself.

As the two subjects are so inseparable they will be considered together.

The alcoholic beverages which are particularly efficient in the production of epilepsy are those which combine some aromatic with the alcohol, and of these absinthe is certainly most remarkable in its power. Horsley has found that when it is injected into the veins of a dog or monkey it produces violent epileptic convulsions, which are eventually followed by death.

In connection with the influence which alcohol exerts in producing epilepsy directly in the drunkard, it may be said that in nearly all such cases there must be lurking somewhere a tendency toward that form of nervous disturbance which breaks forth under the influence of the alcohol circulating in the blood, or is set going by the depraved state of the nervous system, the result of frequent debauch.

Echeverria[1] has analyzed no less than 572 cases of

[1] Journal of Mental Science, Jan., 1881.

alcoholic epilepsy, 307 of whom were males and 265 females. Divided into classes he noted that of these 212 belonged to the middle or upper classes, 306 to the lower classes, while 108 were uneducated, and 37 were outcasts. Dividing all the 572 cases into three classes, he finds :—

1. Two hundred and fifty-seven cases could be traced directly and entirely to alcohol as cause and effect.

2. One hundred and twenty-six cases, in which there was also an associated history of syphilis in 67 and traumatism in 42 as an exciting cause. Of the remaining number of this group ague was given as an additional cause in 2 males, sun-stroke in 9 males, and the excessive use of tobacco in 1 male. Mental anxiety was also an exciting additional cause in 5 cases.

3. One hundred and eighty-nine cases, 92 males and 97 females, in whom alcoholism was the result of the epilepsy, quite as much as the epilepsy was the result of the alcoholism.

Carrying his investigations still further, this observer managed to get a fully completed family history out of 139 cases belonging to the first class and 86 cases belonging to the third division. Of the 139 of the first class, 92 had alcoholism present, alone or with epilepsy, in the parents, while in the 86 cases of division No. 3 a tendency was inherited from insane or epileptic parents or grandparents. In 5 families of persons belonging to this class there were congenital idiots. From these 225 cases he summarizes as follows :—

In 39.33 per cent. there was a direct hereditary tendency to epilepsy or to alcohol and epilepsy. Parental intemperance solely originated the predisposition to epilepsy in 17.30 per cent. Parental intemperance associated with epilepsy or insanity existed in 15.96 per cent.

of the males and 19.24 per cent. of the females, making a total of 17.48 per cent. of the entire number of cases. Parental epilepsy was found in 12.7 per cent. of the males and 15.84 per cent. of the females. If we class together the two preceding kinds of cases we have, respectively, a proportion of 20.10 per cent. in males and 35.47 per cent. in females.

Parental insanity and epilepsy without any history of alcoholism was met with in 4.54 per cent. of the entire number of cases. The reader will note the difference between the percentage of males and females.

Parental intemperance, not ingrafted into epilepsy or insanity without any history of alcohol save in the parents (not grandparents), is 2 per cent. lower in females than in males.

On referring to the aggregate number of cases of intemperance in the parents, irrespectively put together, there is an increase of 15 per cent. in the males over the females. A preponderance of 3.28 per cent. again on the female side when intemperance is associated with heredity, or insanity appears as the hereditary cause; and this difference in favor of the females is almost the same in relation to patients sprung from parents tainted with epilepsy.

Henry Clarke[1] has shown that this preponderance of hereditary epilepsy among females rises to a considerably greater extent as a predisposing cause of epilepsy and crime, the rates being 66.7 per cent. among females, against 38.1 per cent. among males.

Returning to the statistics which we have just left we find that, of the 139 epilepties of the first division with hereditary taint, no less than 64 per cent. of the males and 82.2 per cent. of the females suffered from con-

[1] Brain, January, 1880.

vulsions in childhood, although they did not become epileptic until after 18 years of age, and then in consequence of intemperance. In most of the 86 cases of the third division who had hereditary taint epilepsy was developed in childhood, or before 15 years of age; and it is remarkable that every one of these had suffered from fits in childhood, while in addition 14 had left hemiplegia and 3 right hemiplegia and idiocy. Four had wasting paralysis of one arm, 5 palsy of one leg, 4 Pott's discase of the spine, and 5 faeial palsy. Five had strabismus and 5 deafness and otorrhœa, all dating from infancy.

The chief point in the 67 cases of the second division, complicated with syphilis, is that in 49 of them the fits were followed by palsy. Mental disturbances were more severe in these cases than in those of pure syphilitic brain-lesion.

In the third division there are still 103 cases to be accounted for, in which the cause was ascribed to head injuries and climacteric change.

Echeverria states that he saw the worst cases of alcoholic epilepsy among whisky-drinkers, but he saw none produced by absinthe, probably because it is rarely used in America.

Drouett [1] has collected 445 cases of alcoholism in males, of which 45 were epileptic from alcoholism alone, while in 87 female alcoholics 9 were epileptic. As to the age most common to alcoholic epilepsy, he finds that below 30 years the proportion is 1 in 15; between 30 and 50 years it is 1 in 8.

Moeli [2] has studied the same question in Germany, and reaches the conclusion that alcoholic epilepsy is

[1] Annales Méd. Psychologique, 1875.
[2] Neurologische Centralblatt, 1885.

common, and that from 30 to 40 per cent. of all persons with delirium tremens are epileptic.

M. Hippolyte Martin [1] has also collected interesting facts as to the influence of parental intemperance in the production of epilepsy in children. In 150 cases of insane epilepties at the Salpêtrière he found 83 with such a history. He divides them into two classes, the first comprising 60 cases, or over two-thirds, in which alcoholism in the parents was a certainty, and, second, those in whom such a history was not so clearly defined.

The 60 cases belonging to the first class had 244 brothers and sisters, of whom no less than 48 were afflicted with convulsions in early infancy. One hundred and thirty-two were dead in 1874 and 112 still living, nearly all of them young and nearly all with damaged nervous organizations. Of the second class there were 83 brothers and sisters, of whom 10 were epileptic and 46 still living. These figures do not include the patients themselves, and all the cases were from different families. When we summarize, we find that, in 83 families with 410 children, 108 were epileptic, or more than one-fourth. In 1874, 169 children were dead and 241 living, but 83 of these were epileptic, or more than one-third. Martin also gives the causes of death in the parents, in a number of cases, to show that no hereditary tendency save the alcoholism existed to produce epilepsy. They are as follow:—

Apoplexy, 15.	Suicides, 4.
Heart disease, 5.	Hysteria, 5.
Thoracic disease, 6.	Cancer, 7.
Insanity, 7.	Dropsy (?), 1.
Chagrin (?), 1.	

An interesting case, which, to the author's mind, indicates disease followed by alcoholism rather than

[1] Annales Méd. Psychologique, January, 1879.

alcoholism followed by disease, is one recorded by Bourneville and Baumgarten [1] of a child aged about 4 years. There was marked alcoholism, produced by large quantities of white wine, for about one year before the attack of epilepsy, but at the autopsy there was found a softened condition of the brain and destruction of the cortex, the result of meningo-encephalitis. The cunei were very small, blindness having been present for some months. Microscopical examination revealed sclerosis of the pyramidal ganglion-cells, most marked in the cortex in the occipital region.

Consanguinity.—The effect of consanguinity is believed generally not to be a powerful one, and in this connection it may not be out of place to mention the statistics of Bemiss,[2] who finds that in 31 children born of brother and sister there was 1 epileptic. Of 53 children born of uncle and niece, or aunt and nephew, there was 1 epileptic. Of 234 children born of cousins, themselves the offspring of kindred parents, 4 were epileptic. Of 154 children born of double cousins 2 were epileptic. Of 2778 children born of first cousins 44 were epileptic.

The Influence of Age.—If there is one point upon which all neurologists are agreed, it is the influence of age upon the disease we are considering.

According to the writers whom we have just quoted, when speaking of heredity, three-fourths of all cases of true idiopathic epilepsy begin under 20 years of age, and nearly half of all cases between 10 and 20, the greatest number being at 14, 15, and 16. One-eighth of all cases begin during the first three years of life, but after 20 the number falls very low. The females in these cases exceed the number of males in the first ten years by 6

[1] Le Progrès Méd., 1887.
[2] Medico-Chirurgical Review, July, 1860.

per cent., in the second ten years by 18 per cent., and in the third ten years by 12 per cent. After 30 years the relation is reversed, and the excess of males gradually increases, until, after 60, the malady practically occurs only in males. The following are Gowers's figures in 1450 cases :—

Under 10 years,	422 cases.
" 10 to 19 years,	655 "
" 20 to 39 "	224 "
" 30 to 39 "	87 "
" 40 to 49 "	31 "
" 50 to 59 "	16 "
" 60 to 69 "	4 "
" 70 to 79 "	1 "

In the cases reported by Hamilton the proportion to each age was as follows :—

	Females.	Males.	Total.
Under 10 years,	103	95	198
Between 10 and 20 years,	171	97	268
" 20 and 30 "	145	92	237
" 30 and 50 "	81	136	217
Over 50 years,	11	49	60
	511	469	980

Of 1288 cases collected by nine French authorities, in 486, or over one-third, the disease began between the tenth and twentieth years. Hasse,[1] in the 1000 cases collected by him, found that 75 per cent. of the patients were at the onset under 20 years of age. According to the German, Nothnagel, the great majority of cases occur between 7 and 17 years, not only in hereditary epilepsy, but also in the other forms.

On analysis of the 970 cases collected from literature by the writer, he found that the following results were to be reached :—

[1] Krankheiten des Nervenapparat., Virchow's Handbuch f. Spec. Path. und Therap.

From birth to 10 years,	57 cases.
" 10 years to 20 years,	202 "
" 20 " to 30 "	223 "
" 30 " to 40 "	171 "
" 40 " to 50 "	93 "
Above 50 years,	47 "

The oldest case was 81 years of age.

These results place the largest number of cases between 20 and 30 years. The real boundary of frequency is from 15 to 30 years.

It has been denied that epilepsy, not due to traumatism or brain-tumor, ever occurs in old age, and, while the writer has never seen such a case, there is abundance of clinical testimony as to the falsity of the assertion. Gowers has seen it begin at 65 and 71 years, and Reynolds at 70. Heberden also records a case at 75 years. In Gowers's cases only about 2 per cent. began after the fortieth year, and about 5 per cent. after the thirty-fifth year. A very well known writer on nervous disease, Dr. H. C. Wood, italicizes these words: "An epilepsy which develops after 35 years is not idiopathic, but is due to some organic brain disease, to the abuse of alcohol, reflex irritation, or other causes, which, in some cases, may be so hidden as to be exceedingly difficult of recognition." The same authority also states that epilepsy, in his experience, after 35 years has, in 80 per cent. of the cases, been due to syphilitic brain disease. According to some authors the climacteric period is fertile in the development of epilepsy, but no evidence has been adduced of the proof of this.

Minor Causes.—Passing on from what may be considered the influential factors in all diseases, namely, age and sex, to the various causes, both great and small, which are productive of epilepsy, the writer will briefly go over the minor or more anciently relied upon factors,

and then speak of those which at the present day are more commonly received as really of importance. Among the older observers, Ettmueller and Senac laid great stress upon sexual excesses, but it is extremely doubtful if this is often the cause unless other more powerful agents assist it. In a number of cases, where it is carried to great extremes, some nerve-failure may occur, particularly where there is a tendency to an epileptic condition. In those instances where the sexual excesses take the form of masturbation, the demoralizing influences, coupled with general weakness of intellect, may also predispose to the affection. It has been shown, to be sure, that the first seizure has come on immediately after or during coitus; but this fact has, of course, no connection with causation except when the nervous exaltation of the spinal cord excites reflexly the cranial centres. In respect to the influence of masturbation, Herpin[1] has pointed out that the prevalence of this vice must, to a very large extent, prove it to be a rare causative factor in producing epilepsy. Nothnagel[2] has only seen one such case, and Echeverria calls attention to the fact that masturbation is often resorted to after the person becomes epileptic, although the attacks may have been so long unknown, as in nocturnal epilepsy, as to have escaped attention. Contrariwise, Tissot[3] and some others believe that sexual continence may also result in epilepsy. Herpin[4] is also of this opinion, but limits it to the female sex, while Radcliffe[5] agrees with Tissot. The writer does not think at the present time that much reliance is, or will be, placed on such opinions, and Her-

[1] Du pronostic et du traitement curatif de l'epilepsie. Paris, 1852.
[2] Ziemssen's Encycloped., art. Epilepsy.
[3] Traité de l'Epilepsie. Lausanne, 1790.
[4] Du pronostic et du traitement curatif de l'epilepsie. Paris.
[5] Epilepsy and Allied Disorders. London.

pin's argument against the theory that masturbation is a cause applies equally well here, with even greater force, for the number of females who are not epileptic, but who are sexually continent is, we hope, far beyond the number of female epilepties from every cause. It is probable that these views originally arose by reason of the hysteroid convulsions which may frequently arise from such causes.

Scrofulosis and *rachitis* have been held on high authority as important factors in causing epilepsy by the celebrated English clinician, Anstie [1] who claims that "active hereditary nervousness" is often so produced, resulting ultimately in very much more violent disturbances; and Echeverria also agrees with him to a certain extent, stating that if the parents are pale and badly nourished the children may be epileptic. This evidence is, however, very far from receiving any general confirmation, and there is no reason for believing that epilepsy is more frequently the result of these maladies than many other affections, save in the fact that, by producing asymmetry of skull, cerebral changes may develop. It should be added that Hasse,[2] who was a most careful investigator, also supports the views of Anstie.

Fright produces its effects in either sex probably with equal frequency before puberty, but much more frequently in females than in males after puberty. This becomes the more readily understood when we remember that, as a general rule, emotional causes are important in women, less important in men, with the notable exception that men, being more exposed, more commonly suffer from the effects of prolonged mental anxiety.

[1] Journal of Mental Science, 1873.

[2] Krankheiten des Nervenapparat., Virchow's Handbuch f. Spec. Pathologie and Therapie.

Indeed, where the disease occurs late in life this should be remembered as a cause.

Cases, too, have been recorded by several observers in which epileptic attacks developed late in life as the result of operative procedures against bleeding hæmorrhoids, whereby cerebral congestion resulted.

Of all diseases which may result in epilepsy, either by hereditary taint or by acquirement, syphilis, as everywhere, stands prominently in the foreground. This disease may produce epilepsy, of course, in two ways: either by producing morbid brain-growths or by so influencing the cells of the cerebrum as to cause epileptic convulsions. Under such circumstances the disease very frequently bursts forth at an unexpected moment, and is commonly of great severity. It is during the second stage of syphilis that it commonly comes on, and in such instances we generally find, on inquiry, that the ordinary eruption of syphilide has failed to appear. *Per contra*, we seldom see nervous syphilis where the ravages of the disease have been superficial; that is on the skin.

It is interesting to know that, while simple hereditary epilepsy is more common than the acquired, this is not so with syphilitic epilepsy, for an analysis of a series of cases shows that congenital syphilis is more rarely complicated by epilepsy than the acquired syphilis. Thus, in 618 syphilitic epilepties Echeverria [2] saw only 7 congenital syphilitic epileptics.

One of the curious things which we note as we look back over the literature of this disease is the confidence of each writer that he has discovered the great and only provoking cause of epilepsy. In some instances this

[1] True, Lyon Méd., xlviii, 1885.
[2] Journal of Mental Science, July, 1880, p. 165.

really becomes amusing were it not that the subject is too important to be laughed at.

Thus, Laségue[1] lays the greatest stress upon cranial malformations as a cause of this disease, going so far as to assert that epilepsies not dependent on osseous troubles are not epilepsies at all. Indeed, Laségue is beyond patience in his arbitrary method of statement, for it will be remembered that, when speaking of his work, and that of Garel, under the subject of symptoms, it was shown that such investigations as those on which he based his claim were at least open to great fallacies. Laségue, further, asserts that all the convulsions are identical; that is, never hereditary, and that the attacks are always in the morning if the disease be typical. Where cranial malformations are a cause they probably exert all their influence before the eighteenth year at the latest.

Reflex Epilepsy.—An exceedingly important question has arisen among physicians as to the character of the so-called "reflex epilepsies," some claiming that they are no more a part of true epilepsy than are those of toxæmia or kindred conditions. Perhaps no more forcible upholder of this belief can be brought forward than Reynolds,[2] who vehemently denies that they are a variety of the true epilepsies as recognized at the present day. It will be remembered that the term "reflex epilepsy" is used to signify a convulsive condition in every way similar to the ordinary epileptic convulsion, but which arises by reason of nervous irritation occurring not primarily in the higher motor centres, but in the peripheral nervous apparatus, such as that caused by the involvement of certain nerve-filaments in a cicatrix,

[1] Annales Méd. Psychologique, September, 1877.
[2] Epilepsy. London, 1861.

either new or old, or by an inflamed prepuce or bowel. It should also be kept in mind that the term "reflex epilepsy" is only used to designate a condition of the nervous system in which repeated convulsive attacks have occurred for a long period of time, and does not, as generally employed, signify that form of nervous disturbance shown occasionally during the eruption of a tooth in a child. Russell Reynolds's[1] position is defined by his own words: "Epilepsy should be regarded as an *idiopathic* disease, *i.e.*, a morbus *per se*, which is to be distinguished from eccentric convulsions, from toxæmic convulsions, from the convulsions occurring in connection with organic changes in the cerebro-spinal nervous system, and from every other known and recognizable disease." Notwithstanding this assertion, the same author a little farther on is forced to acknowledge that reflex irritations may occasionally not only produce simple convulsions, but even true epilepsy. He defines his position still more clearly by pointing out that in "reflex epilepsy" "a condition of increased irritability in the reflex centre" has been set up, whereas in the true disorder this morbid condition "has acquired an existence of its own, and the exalted irritability thenceforth depends upon an altered nutrition, which continues even after the removal of the original eccentric source of irritation. It will at once be seen that the border-line between these two conditions is very ill defined pathologically, and the writer hopes elsewhere to show that a peripheral lesion may cause just such central changes as Reynolds believes to be necessary for the presence of real epilepsy. It must constantly be borne in mind that we are discussing in epilepsy a chain of symptoms, expressing some more or less tangible nervous change,

[1] *Loc. cit.*, p. 33.

which in some of its forms is as yet unknown to us. The whole weight of Reynolds's opinion virtually rests upon the ability of the medical attendant to demonstrate some irritation, and the diagnosis is one of epilepsy, or convulsions of another type, according to his capability of judgment. The author quite agrees with Nothnagel [1] that this theory cannot be accepted simply because, in the light of our present knowledge as to reflex degenerations, we cannot deny their occurrence, at least under other circumstances, although we have not as yet microscopical knowledge in this particular instance. The laws of reflex action, so ably put forth by Pflüger, and which are universally received, fully explain how these irritations may result. Further than this, the mere temporary irritability of a cicatrix does not throw the patient into convulsion after convulsion immediately, but the convulsive condition having once been begun by such a cause may be brought back even when no irritation longer exists, of an acute form, by some central disturbing mental condition entirely separated from the periphery. The law may be laid down as an almost unvarying one that every nervous act is followed by other similar nervous acts, rapidly or slowly, according to the severity and frequency of the first acts, and if this predisposition be once set up the subsequent acts may readily be excited by agents which primarily would have produced no effect.

This has been remarked upon by Féré,[2] who states that neurotic children may have an ordinary convulsive attack, which, when repeated, may finally become true epilepsy, and that the eclampsia of scarlet fever may end in a like manner. Féré even believes that the eclampsia of pregnancy may so result in some cases,

[1] *Loc. cit.* [2] Arch. de Neurologie, 1885.

and he speaks of "epilepsies eclamptiques" as a variety of the disease in which a single convulsion comes on as the result of great excitement, and which may become permanent or transient.

Again, it is to be pointed out that the epileptic seizure often has no relation whatsoever to the degree of irritation present at the time. To be sure, this binds one to the doctrine that in every instance of reflex epilepsy organic central changes take place, and if one assents to such a belief, then it is asked why the removal of the peripheral irritation in many cases cures the disease. The answer is that in these cases the peripheral lesion has not existed long enough or been violent enough to produce such changes in the central nervous system as to place it beyond the realm of recovery, and that in many instances where the removal of an irritation fails to produce a cure it is the lateness of the operation that permits of too great central change to be remedied by nature's power. Very commonly, the removal of the irritation is not followed by an immediate and complete remission of the seizures, but they occur at more and more irregular intervals until they cease, showing that a central change or tendency to convulsive explosions must have been set up. The writer is exceedingly desirous that the fact already stated be remembered, namely, that the very occurrence of one nervous act predisposes to a similar act at some future period.

It is now many years since the possibility of adherent prepuce producing epilepsy was first brought forward, and there can be no doubt of the truth of the assertion that the removal of this redundant skin and mucous membrane is accompanied by a cure in some cases. It has been claimed by some writers, however, that the convulsions in all cases have been hysterical in character,

and have been cured by the shock of the operation rather than by the removal of a peripheral irritation. There can be no doubt that in this belief there is a certain element of truth, even though the convulsion be not purely hysteroid in type, but it is far more absurd to ascribe the shock as the chief means of cure than to deny entirely that it has any influence. In the first place, the removal of an adherent or inflamed prepuce produces benefit in cases where the slightest hysterical symptoms are absent, and also in cases where the entire atmosphere of the case forbids such a possibility. Every one has seen cases of young children of no more than a year, in whom the presence of true hysteria was not to be thought of, pass day by day into a greater state of nervous strain, which finally ruptures into a well-defined epileptic attack, and in whom the removal of the prepuce, when it has been found inflamed, produces a complete cure, either when the child has suffered till it is 2 or 3 years of age or as soon as the physician has been quick enough to perceive the cause of the trouble. The writer has himself relieved a case as early as the eleventh month by such an operation. The same results are frequently reached by similar operations upon persons of much more advanced age, particularly if the exciting cause is recognized so early that the disease has not become chronic. Every one will agree that the relief obtained by circumcision is in direct proportion to the youth of the sufferer and (if one may use such an expression) the youth of the disease.

It is also fortunately true that such seizures are most commonly among young boys below puberty, while they are exceedingly rare among men for the reasons mentioned elsewhere. In young children, also, smegma and small quantities of urine frequently are allowed to re-

main unnoticed behind the foreskin, which, in the adult who is careless, soon call his attention to the part by reason of the inflammation produced. It should be a routine practice with every practitioner to examine carefully the penis of every male child brought to him with epilepsy, particularly during early youth.

Closely allied to the reflex epilepsy of preputial irritation is that supposed to arise in females from similar irritation of the clitoris or vagina. Necessarily, such cases are exceedingly rare among children, but become more common as the period of puberty is approached,—in some cases by reason of the changes occurring in the parts, in others by the carrying out of pernicious practices whereby mechanical irritation results in a chronic inflammation. It will be seen, therefore, that reflex epilepsy from the clitoris is more common about the ages of 10 to 15 than that produced in boys about the age of 5 or 6 by penile irritation.

It also becomes equally evident that in the girls the hysterical character of the seizures may be much more strongly developed, owing to the age, sex, and original cause, since in masturbating girls the hysterical condition is either present as a cause or result of the self-abuse, in many cases. As he writes a case comes before the author of a girl of 18 years, of a typical hysteroid appearance, but well developed mentally and physically, in whom the tendency for self-abuse was so strong that, after all other measures had been tried, the operation of oöphorectomy was performed with the hope of curing the desire, which, in its frequent gratification, produced such an increase in the hysterical seizures as to border on true epileptic convulsions. The operation did not, however, remedy the evil; at least, while she remained under observation.

A condition of the clitoris which is very rare, but which has been supposed to produce epilepsy, is elongation of that organ to such an extent that it is constantly in contact with the patient's clothes, and gives rise to sexual desires which cannot be satisfied, or becomes excoriated by the rubbing of the clothes wet with vaginal discharges, even though they be of normal character. For the irritation of masturbation, or that last mentioned, clitoridectomy has been proposed and tried to a limited extent; at least, in this country. The cases have been so few that it is scarcely possible to draw conclusions from the results obtained, even when the cases reported from all nations are sought out.

The writer has already referred to the reflex irritation caused by vaginal irritation due to the escape of the *oxyuris vermicularis* from the rectum into the vagina. In many cases the local inflammation is so severe that attention is at once called to it, but in other instances the signs are by no means connected with the seat of the disease, and consequently pass by unnoticed. It may be laid down as a fact that in all cases in girls in which epilepsy of unknown cause develops the vagina should be examined, and if the history is one showing the presence of worms this becomes most essentially a part of the search after health.

Perhaps no form of reflex peripheral irritation producing epileptiform seizures has been so commonly sought after and relied upon as that due to intestinal indigestion or intestinal worms. The first has rather been looked upon as the cause of isolated convulsions occurring in nervous children; the second has, while partaking in this belief, been generally considered to produce more frequent discharges of nerve-force, or, in other words, repeated seizures. That indigestion may

No.	Age.	Married.	Single.	Masturbator.	Condition of Clitoris.	Duration of Epilepsy.	Result.
1	26	Not stated.	.....	Not stated.	Not stated.	22 years.	Benefited, but disease returned in 1 month.
2	41	.....	.....	Not stated.	Not stated.	12 years.	Benefited.
3	16	.....	Single.	Yes.	Not stated.	9 years.	Slight benefit.
4	Not stated.	.....	Single.	Not stated.	Not stated.	9 years.	Cured.
5	41	.....	Not stated.	Not stated.	Not stated.	15 years.	Cured.
6	25	.....	.....	.....	.....	Many years.	Cured.
7	31	Married.	.....	.....	.....	Many years.	Cured.
8	44	Married.	.....	Not stated.	.....	4 years.	Cured.
9	33	Married.	.....	Not stated.	Swollen and inflamed.	6 years.	Cured.
10	24	.....	Single.	Not stated.	Not stated.	3½ years.	Cured.
11	41	.....	Single.	Not stated.	Irritable.	12 years.	Cured.
12	28	.....	Single.	Not stated.	Not stated.	11 years.	Cured.
13	21	Married.	.....	Not stated.	Not stated.	10 or 11 years.	Cured.
14	20	Not stated.	.....	Not stated.	Not stated.	2 years.	Cured.
15	21	.....	Single.	Not stated.	Not stated.	3½ years.	Cured.
16	17	Not stated.	.....	Not stated.	Not stated.	1 year.	Cured.
17	16	.....	Single.	Not stated.	Irritable.	2½ years.	Soulagement.
18	44	.....	Single.	Not stated.	Not stated.	30 years.	Cured.
19	19	.....	Single.	Not stated.	Hypertrophied.	7 years.	Relief.

set up either isolated or frequent attaeks is without doubt true, and it is even possible that this may give rise to an epilepsy lasting a life-time, for, as has already been said, every nervous act is followed by its duplicate, if sufficient cause exists. The following case from the writer's note-book illustrates this very well :—

A. E., girl, aged 7 years, was brought to me by her mother, who stated that she suffered from epilepsy of an irregular type, which occurred seemingly without cause.

During the few weeks preceding her visit convulsions had occurred as commonly as three or four times a week, which, from the description of the mother, were evidently epileptic in form. Previous to this no seizures had occurred for some months, the first attaek having taken place at about the age of 4 years. The child was put upon the routine treatment of bromide of potassium with only slight amelioration of her symptoms. After a few days the mother returned, and it was discovered that the father had of late been in the habit of feeding the child, giving it any of the food on his own plate that the child desired. It was also discovered that corned beef was frequently eaten by the father and as commonly by the patient, and that the nervous disturbance generally followed its ingestion. For the sake of convincing the parents that this was the cause of the trouble, they were ordered to feed her for two weeks on only bread and milk and corresponding articles of diet, and after that to give her an ordinary amount of corned beef. The result was another convulsion. Without occupying more space by a further account of the case, it may be added that recovery with no return of the fits occurred upon careful regulation of the diet and attention to the digestion, the only fit following being one two months

later, when, owing to the absence of the mother, a relative gave the child some indigestible food. The case just cited is but an evidence of the fact that, apparently, true epilepsy may be produced by very easily removed causes.

The convulsions of young children occurring under conditions of indigestion are, while epileptiform in character, scarcely true epilepsy, and their treatment may resolve itself simply into the prevention of the ingestion of harmful matters and the use of nervous sedatives when the crisis is at hand.

Ovarian irritation has been accused of exciting true epilepsy, and in a limited number of cases it is, of course, a real cause, which can only be removed by a radical operation.

Still another cause has recently been prominently brought forward in America by Dr. Stevens, of New York, and Dr. A. H. Thomas, of Philadelphia, viz., epilepsy the result of strain and consequent reflex irritation of the muscles of the eye, and they have devised an operation closely allied to that ordinarily used in the connection of strabismus with asserted good results. Rather curiously, the writer has himself seen a patient on the Continent who, although operated on by Dr. Thomas in America, experienced relief for only a few months, and is now almost as frequently and severely attacked as before.

Disease of the external or middle ear may in some cases be associated with reflex epilepsy, entirely independent of any direct involvement of the brain itself. Thus, Moos[1] reports a case of middle-ear disease resulting in epileptic seizures, and traces the inflammatory irritation along the tympanic plexus, and thence to the

[1] Allgem. Zeitschrift f. Psychiatrie, xxxii, 5.

brain. A large number of such instances are recorded elsewhere by other writers.

Quite an elaborate research on the relation of epilepsy to ear disease, when the latter acts as a causative factor, has been carried out by Boucheron,[1] and, while it is far too long to be quoted here to any extent, the results are sufficiently interesting to be recorded.

1. Epileptic attacks occur in infants during the evolution of the auricular affection which results in deaf-mutism.

2. They occur in childhood during the course of various affections of the ear.

3. In adult life they occur from the same causes, or in other cases the trouble may reside in stoppage of the Eustachian tube.

Boucheron also points out that epilepsy may be produced in dogs and cats by the presence of foreign bodies in the ears.

A research by Ormerod[2] has also reached somewhat similar conclusions, for he tested the ears of 200 epileptic patients, and found that 31 of them had a previous history of suppurative otitis, past or present. As a counter-study he examined 100 patients with ear disease for epilepsy, and found 7 cases. It will be seen that this percentage is very high, for Niemeyer[3] placed the proportion of epileptics to the population of a country at 7 per 1000, which is also absurdly high, as will be shown later on.

Very much more recently Boucheron[4] presented a paper in which he characterizes the epilepsy of this variety as consecutive to a direct action upon the auditory

[1] Revue de Thér. Méd. Chir., September 1, 1886.
[2] Brain, 1884, p. 37.
[3] Practische Med., art. Epilepsie.
[4] Société Française d'Otologie et de Laryngologie, April 27, 1888.

nerve. He quotes Noquet, who observed a deaf-mute affected with tinnitus and epileptic attacks, who was cured by Politzer's inflation of the tympanum, and he states that certain epilepsies can originate from compression of the cerebral convolution where the auditory nerve originates.

Galezowski [1] has reported a case of epilepsy due to the reflex irritation produced by inflammation of the stump of an optic nerve after the eye had been removed.

One could go on mentioning peripheral irritations producing epilepsy almost indefinitely, but the writer will call attention to only one or two. Brubaker [2] has reported 18 cases of epilepsy purely due to the presence of carious teeth, and Schwartzkopff [3] has reported a very interesting instance. Liebert records 3 cases of this character, in every one of which the aura was remarkable in that it consisted in a cramp of the muscles of the tongue or involuntary movements of that organ. Each case was cured by the removal of a carious tooth. Baly,[4] Booth,[5] and Hamilton [6] have done likewise.

Ross [7] reports a case where gall-stones produced the disease reflexly.

Some of the other peripheral causes are stenosis of the uterine cervix, uterine disease of various forms, and ovarian disease. Cases have also been reported as caused by stone in the bladder, [8] and a very peculiar case is recorded in which the irritation of a misapplied hernial truss caused epilepsy.

[1] Revue de Thér. Méd. Chir., 1886.
[2] Med. and Surg. Rep., Philadelphia, 1888.
[3] Deutsches Monatshefte f. Zahnheilk., Heft 3, September, 1885.
[4] London Med. Gazette, 1851, xlviii, 534.
[5] Amer. Jour. Med. Sciences, 1870, n. s., lix, p. 278.
[6] *Loc. cit.* [7] Nervous Diseases, London.
[8] Edinburgh Med. and Surg. Jour., 1868-69, xiv, p. 140, and Arch. Sci. and Pract. Med. and Surg., 1873, p. 1360.

One of the best evidences that we have of the influence of peripheral irritation in provoking epilepsy is presented in the reports of cases where a close examination of a given nerve showed it to be diseased, and where its removal was followed by cure. Thus, Lande[1] has recorded a case following injury to the right median nerve, and Short[2] has reported an epilepsy produced by the growth of a neuroma. Not only has Brown-Séquard shown, as will be pointed out farther on, that injury to the sciatic nerve may produce epilepsy, but Billroth,[3] Garnier,[4] and Raymond[5] have shown that in similar cases in man excision of the injured nerve produces relief. Briand[6] has also recorded a case of great interest in this connection, namely, that of a man who was struck by a fragment of an exploded shell across the buttocks in the battle of Mans in 1871, the sciatic nerves on both sides being injured. Eight months after the injury epileptic fits came on, which constantly recurred. Several cases of a somewhat similar type have been recorded by Billroth[7] and Shaefer[8].

A form of peripheral lesion producing epilepsy, probably not by reflex action, is that recorded by Schultz,[9] where prolonged compression of the jugular vein caused the disease, or in those cases where enlarged cervical glands bring about a similar result.

Barometric Changes.—The influence of barometric changes upon epilepsy has been frequently studied,

[1] Mém. et Bull. Soc. de Méd. et Chir. de Bordeaux, i, p. 56, 1878.
[2] Medical Essays and Observations Soc. Edinburgh, iv, 416, 1737.
[3] Archiv f. klin. Chirurgie, xiii, 379, 1872.
[4] L'Union Méd. de Paris, 3 s., xiii, 65, 1872.
[5] Revue Méd. de Timoges, iii, p. 102, 1869-72.
[6] Bulletin de la Soc. d'Méd. d'Angers Ann., lxxvii, p. 121.
[7] Langenbeck's Arch., Bd. XIII, 1871.
[8] Aertzl. Intellingenzblat, 1871.
[9] Correspondensblatt Deutsches Gesellschaft f. Psych., ii, p. 35, 1855.

always with very contradictory results, but Delasiauve,[1] by a long series of studies, states that the attacks are more common during the season of the year when the prevailing winds are from the northwest, north, and southwest,—a rather remarkable conclusion. The conclusions reached by Leuret[2] are, that the season of the year has little influence, and that if any such influence exists it is during the autumn and winter, the fits being, perhaps, a little farther apart in summer and spring.

Not only are the statements of this writer not generally received, but an immense mass of testimony shows epilepsy to be so wide-spread that no particular winds can favor it. There is, probably, no disease under heaven which is more ubiquitous than epilepsy. It is in Greenland,[3] in Iceland, in India, and in Africa. In Sweden and Norway it also appears.[4] Even the far-off islands of the Pacific see the disease.[5]

Toxæmic Epilepsy.—*Uræmia.*—Nothing particular is here to be said in regard to this variety of convulsive condition, for the matter will, perforce, have to be discussed when the subject of diagnosis is considered.

Exanthematous Fevers.—One of the causes which, in a certain number of cases, certainly acts in producing epilepsy is that of these febrile disorders. Every writer of any wide experience, writing on epilepsy, mentions this fact in giving the subject a general discussion, but very few statistics have actually been collected in regard to the frequency with which they thus act. The Englishman, Gowers, found that in 35 cases of epilepsy not

[1] Traité de l'Epilepsie. Paris.

[2] Arch. gén. de méd., mai, 1843.

[3] Cranz, Historie von Grönland, Barby, i, 189, 1770; and Lange, Bemaerkn. om Grönlands Sygdomsforhold, Kfobenk, p. 42, 1864.

[4] Pontoppidan Historie von Norwegen. Copenhagen, 1754.

[5] Hirsche's Handbuch f. Geograph. und Histor. Medicine.

less than 19 were due to scarlet fever and 9 measles; while in America Hamilton has seen, in the 930 cases collected by him, but 23 cases of epilepsy due to scarlet fever and 6 due to cerebro-spinal meningitis.

As Gowers saw no less than 1450 cases, but only found 35 due to exanthemata in the entire number, and Hamilton only saw 29 cases of the same character in 980 cases, it is at once evident that these causes must be very rare as compared to some of the others.

These fevers may reach the result in three ways in producing epilepsy: either they light up into action cells already slightly deranged, or produce this derangement primarily; or, again, they give rise to certain more gross pathological changes, such as abscess or tumor.

It has been said by some that the convulsions which find their way out into open manifestation during or after the disease has spent itself are generally due to the uræmia produced by renal changes, and that this is supported by the fact that scarlet fever is more commonly followed by renal change and epilepsy than are the other exanthemata. Aside from the fallacy of such methods of reasoning, it is at once evident that where the epilepsy is continued for a long time it cannot be due to the accumulation of effete products in the blood, and also by the fact that these cases show no signs of renal disease during life, nor does the post-mortem examination discover any change from the normal state. Then, too, changes are found sometimes in the brain which show that the hand of the disease has been felt there. Besides all this we find measles, which is rarely complicated by renal trouble, standing next to scarlet fever as the most common cause of epilepsy among the eruptive diseases of childhood, associated with

fever, and it is by no means rare to see cases of measles followed by gross morbid lesions in the cranium.

Thus, in one case, which occurred in the practice of a brother practitioner, a child in perfect health and with an enviable family history, became absolutely idiotic after an attack of measles, and remains so till this day (2 years). Everything has been done that can be attempted, and a consultation of some of the most noted neurologists living has resulted in a diagnosis of a large arachnoid cyst. No epileptic symptoms have developed, but it is believed that this depends upon the area involved rather than the smallness of the lesion.

The writer has not found any cases of epilepsy directly traceable to *diphtheria*, although he has seen very large numbers of cases with other nervous diseases from this cause, such as paralysis and kindred affections; and, while he cannot deduce any statistics directly on this point, he is sure the fact remains that, though diphtheria plays havoc in the nervous protoplasm of the body very frequently, it rarely seems to cause the changes necessary for the production of epileptic spasms.

The convulsions so often seen in those children about to be stricken down by an acute disease, and which may be regarded as prodromata, are often quite as epileptiform in nature as any others with which we may come in contact, but they may be only tetanic in character. They are due, so far as we know, rather to functional disturbance than organic change.

Lead.—A cause of epilepsy w ch is rarely seen is chronic poisoning by lead resulting in the condition known as *encephalopathia saturnina*, or saturnine cerebritis. It may be said that it is rare because its chief student, the well-known Monsieur E. Tanquerel, has only seen 6 cases of saturnine epilepsy in 72 cases of lead

poisoning. With some practitioners its occurrence may never be seen, for it generally comes on in artisans working in the metal, and rarely, or never, does it complicate the milder forms of poisoning commonly observed. Tanquerel and others have noted that, as a general rule, the period of exposure prior to the first paroxysm is one year, but it varies from a few months to many years. Women suffer, naturally, less frequently than men, owing to their occupation. It has also been proved that the ages most commonly affected range from 20 to 50 years, probably due to the fact that these persons more generally work in lead than older or younger men.

The manner in which this metallic poison produces epilepsy is only known in its pathological aspect, and will be spoken of more fully when considering the pathology of the disease, only mentioning it at this point lest the reader should think it had been overlooked. The same may be said of arsenic and mercury, as well as some other poisons of the same class.

Epileptic crises sometimes occur in rheumatic or gouty persons, seemingly as a result of an excess of the diathetic poison present in the blood. This cause is well recognized, and the five cases reported by Teissier[1] may here be mentioned.

Thus far the writer has spoken of the so-called idiopathic epilepsy, Jacksonian epilepsy, reflex epilepsy, and toxæmic epilepsy, and he will now call the reader's attention to a form of the disease which can scarcely be called epilepsy without some qualification, since, while resembling the ordinary seizure in every respect, it requires a directly-acting exciting cause on each occasion. Nevertheless, as it is closely allied to the epilepsies due to chronic peripheral irritations, this seems the most appro-

[1] Lyon Méd., t. xlviii, 1885.

priate point at which to speak of it. He refers to the so-called *Epilepsie Pleuritique.*

The first person to call attention to this form of convulsive attack was Maurice Raynaud, of Paris, in 1875, and since that time quite a number of well-known clinicians have seen and reported cases. The direct cause is the injection into the pleural cavity of some medicinal agent, such as iodine, chloral, or carbolic acid. That the nervous disturbance has not been due to absorption has been proved again and again by direct experiment, and there can be no doubt that the cause is the sudden peripheral irritation which results in an impulse which, being received by some centres peculiarly situated, results in an epileptic seizure. The convulsions are often very severe indeed, and in one or two cases have resulted in death.

Cardiac epilepsy depends upon almost unknown causes, and is probably due entirely to disorder of the nervous apparatus governing the heart, either in the higher centres or in the collateral anastomoses of the circulatory nervous apparatus. According to both Blondeau[1] and Stokes[2] the primary lesion depends rather upon a change in the heart itself, namely, fatty degeneration, but Halberton has seen a similar case depending entirely on an injury to the neck resulting in lesions in the cranial contents. It is evident, however, that the degenerations seen in the cardiac muscle by Blondeau and Stokes were not the real lesions producing the disease, except perhaps indirectly and in a reflex manner, and that, while the symptoms were the same in each case, the lesions recorded by Halberton were more probably the true ones. It is probable, too,

[1] Etudes cliniques sur le Pouls lent permanent. Paris, 1879.
[2] Diseases of the Heart and Arteries. London.

that those cases seen by Stokes had attacks separated by a very narrow border-line from those of ordinary syncope, for he states that one of them could always abort a paroxysm by getting down upon his hands and knees and placing his head downward. It should be remembered, on the other hand, that the relief afforded to the heart-muscle and its nerves by this saving of its strength may have been akin to the use of a ligature in preventing the progress of an aura, or by the increased supply of blood to the brain have overcome the tendency to convulsive discharge.

Having gone over in a thorough manner both the near and remote causes of epilepsy, let us pass on to the consideration of the causes of some of the symptoms individually found to make up the entire diseased manifestations. By far the most important of these is the convulsion itself, which from its character we know must arise from some portion of the nervous system above the spinal cord, if by no other means than the fact which is universally recognized, namely, that clonic, or, as they have been called, epileptiform, spasmodic movements are always due to a perverted cerebral action, while the tonic spasm depends upon a spinal discharge of impulses. It may be asked, Why is it that, if all the perversion of function resides in the cerebrum, there is at the first part of the fit a distinct stage in which the muscles affected are in a condition of tetanic contraction? Every one acknowledges that the great cause of all convulsions is the sudden liberation or explosion of nerve-force, which sweeps everything before it.

Originating, undoubtedly, in the cerebral motor centres, it passes, in epilepsy, instantly through the great conducting paths in the internal capsules down to the spinal cord, and from here to the muscles affected, which

are thrown into tonic contraction for the moment by the suddenness and excess of the impulse which may have awakened the spinal cells to their automatic action and caused, in addition to the cerebral influence, an impulse which may be said to be truly spinal in character. The centres of the cerebrum do not send out, in all probability, one constant stream of nerve-force in health or disease, and even those of the spinal cord produce tetanic spasm by impulses coming so rapidly one after another that the muscles do not have time to relax. The fact that it has been found possible to produce tetanus in a muscle by rapidly interrupted stimulation, and that if the impulses are diminished in frequency a slight attempt at relaxation is recorded on the revolving drum, proves that this probably occurs when the impulses come from the nervous centres instead of a galvanic battery.

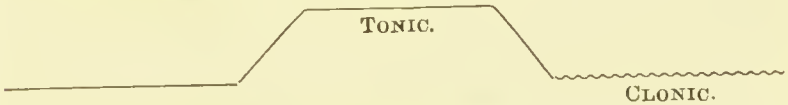
As time elapses after the first impulse has been sent out, the stream of nervous force becomes more interrupted, and consentaneously with this the muscles find time to relax more or less between each impulse. The writer has already stated elsewhere that the last spasm of a fit is often as severe as the first, but this in no way contradicts his argument, for it is perfectly possible that at first the stream flows rapidly because of a plentitude of nerve-power, but later on, as this is lost, moments or pauses must occur in which the cells gather strength. To make this more clear, let the author use a simile[1] :—

[1] Since the writing of these words the views here expressed have received such singular confirmation that these statements are no longer in any way hypothetical, for Horsley has recently found that he can lead off impulses, arising in the brain, from the spinal cord, and by the aid of a capillary electrometer and a photographic apparatus record the impulse. Thus, on producing epileptic movements by galvanizing the cortex in the leg area, he found that both the tonic contraction and the clonic contractions arose in the brain, for the impulse, when led off

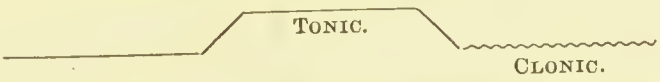
If a general of an army orders several regiments to charge a redoubt, and feels that he has a good reserve behind him, the orders and the resulting charges may follow one another in so rapid a sequence as to be virtually a continuous stream of force, hurling itself on the enemy. As the time goes by, the force of troops in reserve begins to diminish, and, as a consequence, it is not possible for that general to send out so many impulses; but this in no way affects the severity and power in each individual charge, and the force discharged by the twentieth individual regiment may be as great as that of the first, although a period may have preceded it during which the remaining regiments were gathering force for the charge. Indeed, the last charge may surpass in severity the others, in that it may be strengthened by the survivors driven back from the others. The writer believes it is just so with the motor cells of the cerebral cortex. The whole history of the attacks supports this belief, for the first prodromal stage of restlessness so often seen for some days may be said to be the evidence of the massing, not of bodies of troops, but of units of nerve-force, which, as they accumulate, soon run over. The subsequent history of the case bears me out still further; for we often find exhaustion of the motor

from the spinal cord, gave a tracing exactly like that of ordinary epilepsy. Graphically, this may be shown by the following tracing.

That of ordinary epilepsy is thus :—



That of stimulation of cortical areas is thus :—



The first is the muscle tracing of an epileptic fit. The second is a tracing of the nerve-impulse, as shown in the capillary electrometer.

centres evidenced by paralysis or depression of function, and this lasts until sufficient time has elapsed to permit of reparative change.

That the rapid discharge of impulses is always followed by intermittent discharges has been proved to be the case by direct experimentation even when the subject is a normal man. Thus, two American enthusiasts in the study of neurology, Dercum[1] and Parker, found that if they stood with the tips of the fingers just touching the smooth surface of a table when their arms were completely extended as far as possible by an effort of the will, and depended on the brain-centres solely for their steadiness because the surface of the table was touched too lightly to afford support, they found, it is repeated, that in a short time they were unable to keep the arms immovable, and that, if they attempted it, jerkings of the muscles came on, which, when the position was persisted in, eventually spread to the whole body and caused them to fall to the ground convulsed from head to foot, although consciousness was preserved. That the convulsions were *bona fide* in character is certain, for they exhibited these phenomena before the Neurological Society of Philadelphia. The writer has spoken several times of the post-convulsive paralysis as due to exhaustion, and, aside from the fact that all living matter becomes exhausted by excessive action, and that certain areas of the body are often paralyzed after being most convulsed, direct experimental evidence of a very simple character is at hand.

In a very interesting research carried out by the writer's late friend and colleague, Dr. N. A. Randolph, he employed a series of convicts, under his supervision, in using on one day the fore-finger of the right hand to

[1] Proceedings of the Neurological Society of Philadelphia, 1884.

depress a small key arranged on a lever so as to record the number of depressions, and he found that after a certain time the men were unable to go on, even though bribes of luxuries were made and prizes offered to the man who reached the highest number of depressions. It could be seen that the will to move the finger passed in the man's brain to the fore-finger centre but that arriving there it found that centre too exhausted to obey. It was also proved that the exhaustion of the centre for the right fore-finger partially destroyed the power of the centre for the left fore-finger, for my friend first made a series of control experiments and determined, let us say, that the maximum number of depressions of the key made by a right-handed man with his right fore-finger was 250, and that the maximum number made by the left fore-finger was 200. If the man attempted to depress the key 250 times with his right fore-finger, having previously depressed the key with his left fore-finger till its centre was exhausted (200 times), he was unable to do more than 200 depressions instead of 250, or if 250 depressions were first made with the right fore-finger only 150 could be made with the left fore-finger. These results, of course, prove that the two centres are not absolutely independent of one another.

The causation of unilateral or monobrachial epilepsies will be discussed under the question of the pathology of epilepsy.

The writer has already, when describing the symptoms of an attack, stated that the cause of the dilatation of the pupils is the asphyxia, and also that the primary pallor in an attack may be due to fear or vaso-motor disturbance, while the cyanosis following it is from the asphyxia which results from interference with the respiratory movements.

Pathology.—The author has already so often insisted upon the fact that our knowledge of this disease is not what it should be that he is almost ashamed to speak of it here once more, but of all the sections of this essay this chapter impresses one with the wisdom of offering a prize for the best discussion of our present amount of knowledge concerning epilepsy, since, up to the present time, no essay has appeared which gives the results of the labors of investigators in this disease within the past twenty years.

At the very beginning of the study we are forced to acknowledge the impeachment that histology and anatomy fail to give us any insight whatever into the cause of the nervous disturbances of idiopathic epilepsy. All we can do is to accept the generally-received doctrine that the convulsions are the result of an explosion of nerve-force in the higher cortical or subcortical brain-centres, and that the auræ and like preliminary manifestations are in many cases of the same origin

The question of the physiology, or rather the pathology, of epilepsy has attracted a very great array of investigators ever since the dawn of the present medical era, but it is only those of the last fifty years who have pushed our knowledge forward to any great degree, and it is probable that in the last ten years more has been done than in the preceding forty. While all these students have aided the subject, more or less, there still remains much for them to do, though at the present time the study of the functions of the nervous system, particularly the brain, is being carried on with the greatest vigor and accuracy, and we can have no reason to doubt but that a speedy enlightenment of our ideas will soon take place.

The writer has called attention more than once to

the results of Brown-Séquard with a passing remark, but in this portion of the essay he desires to give as much of his results, and those of others, as will give a clear idea of the influence which experimentation has had upon our knowledge of this disorder. For many years this world-widely known investigator has published numerous papers bearing upon this matter, the references to the chief of which are appended.[1] He found that section of one or both sciatic nerves, wounding of the medulla oblongata, or of the cerebral peduncles or quadrigeminal bodies, produced epileptiform paroxysms at varying lengths of time after the infliction of the injury. He also determined that section of one lateral half of the spinal cord produced similar convulsive disturbances. Section of the entire cord may so result, or even a single puncture is often sufficient to bring on the movements. It was also found that these injuries to the cord were more certainly epileptogenic in some regions than in others, as, for example, between the eighth dorsal and second lumbar vertebræ. After the elapse of from twelve to seventy-one days it was noticed that certain muscles were excessively irritable, and soon a general epileptic state developed. The first attack may be spontaneous, but, as is well known now to every one, succeeding attacks can be produced by touching or pinching some portion of the skin of the animal,—a very common situation for this zone being at the angle of the jaw, which, therefore, received from Brown-Séquard the title of the "epileptogenous zone." This zone is always on the side on which the injury is received unless that

[1] Researches on Epilepsy; its Artificial Production in Animals, and its Etiology, Nature, and Treatment. Boston and Philadelphia, 1857.

Journal de physiologie de l'homme, vols. i and iii, 1858 and 1860, and in vols. i to iv Arch. de Physiologie normale et pathologique (Brown-Séquard, Charcot, and Vulpian), 1868–1872.

injury be to the cerebral crus, when it occurs on the opposite side. Even more remarkable is the condition of this area as regards sensibility, for, while it is to some degree lacking in sensibility, the slightest irritation of it may produce the convulsion. After the epileptic paroxysms have lasted for months or for years they subside, and the anæsthesia of the "epileptogenous zone" also decreases *pari passu* with the subsidence of the spasms. A most extraordinary fact is, however, that the young of such animals—always guinea-pigs—are often spontaneously epileptic, which is doubly interesting on account of its scientific and clinical interest.

These zones have not been commonly found in man. Schnee had a case of a woman who suffered from a scalp wound, and pressure on the resulting cicatrix always caused a fit. Neftel[1] saw an epileptogenic zone develop over the right eye. The writer has already called attention to the case recorded by Briand,[2] of injury to both sciatic nerves in man followed by epileptic attacks.

These results did not only occur in the animals under the care of Brown-Séquard, but many men, all over the world, have proved their truth, notably Schiff[3] in Geneva, and Westphal[4] and Nothnagel,[5] the latter seeing instances in which the attacks came on, in as short a time as thirty-six hours after the injury, of such a severe character as to cause the death of a strong, healthy animal.

Schiff obtained convulsions in from three to four days. So far, convulsions have not been produced in cats and dogs and rabbits with the same degree of suc-

[1] Arch. f. Psychiatrie, Bd. VII, 1877.

[2] Bulletin de la Soc. de méd. d'Angers. Ann. lxxvii, p. 121. (See page 117 in this essay.)

[3] Lehrbuch der Physiologie, 1858–59.

[4] Berliner klin. Wochenschrift, 1873, No. 38.

[5] Ziemssen's Encyelop., art. Epilepsy.

cess as they have been in guinea-pigs, but Schiff has produced them in dogs without any loss of consciousness, and the writer has thrown guinea-pigs into such conditions a number of times, and always with results exactly similar to those given above.

The researches of Westphal were somewhat different in method, but similar in result, with those of the other observers. He found that tapping light blows on the head brought about convulsions immediately, and after the elapse of a few weeks epileptic convulsions of apparently spontaneous origin came on, and that there existed an "epileptogenous zone." Coma frequently complicated these fits, however, and post-mortem examination showed small hæmorrhages into the spinal cord and medulla oblongata.

As is well known to all the readers of this essay, epilepsy was at one time very generally believed to be due to a disorder of the circulation at the base of the brain, and, acting on this belief as long ago as 1836, Cooper,[1] of England, attempted to show, by some imperfect and defective experiments on the lower animals, that this was the cause. His belief was most strongly seconded by two others of his countrymen, namely, Marshall Hall and Travers,[2] who pointed out the resemblance between true epilepsy and the convulsions of cerebral anæmia from hæmorrhage.

Later than this Küssmaul[3] and his colleague, Tenner, performed a very large number of similar, but more careful, experiments, with the result of confirming the assertion that epileptiform convulsions could be pro-

[1] Guy's Hospital Reports, vol. i. London, 1836.

[2] New Sydenham Society's Transactions, 1859.

[3] Untersuchungen über Ursprung und Wesen der fallsuchtigen Zuchungen bei der Verblutung so wie Fallsucht überhaupt. Frankfurt, 1857.

dueed by sudden eerebral anæmia. This has been eonfirmed by many other investigators, notably Wood,[1] of Ameriea, who has found that the eonvulsions produced by most of the drugs which aet as eardiac depressants, when taken in toxie amounts, depend on cerebral anæmia for their origin. Nothnagel as well as Küssmaul and Tenner have endeavored to produce similar results by galvanization of the eervieal sympathetie nerves, but Nothnagel[2] failed to get any convulsive result, and Küssmaul and Tenner only sueeeeded onee.

It is as easy to explain why Nothnagel reaehed no results as it is difficult to discover in what manner the others sueeeeded but once, for the vasomotor system of the eerebrum is not governed by the fibres of these nerves at all. Nothnagel has, however, galvanized peripheral sensory nerves in order to produee vasomotor spasm and convulsions with sueeess, aeeording to his report, and Krauspe[3] has by a very careful and exeellent series of experiments eonfirmed these results. One eannot help thinking, however, that the eonvulsions oeeurring under sueh eireumstanees were not due to the vasomotor spasm produeed by the stimulation of a peripheral sensory nerve, for several reasons. In the first plaee, the writer has galvanized the seiatie nerve again and again, with all degrees of strength of eurrent, and never seen any eonvulsive movement, but, as he did not attempt to produee eonvulsions, but to determine the integrity of the vasomotor system, he may not have eontinued the stimulation long enough to ause spasmodie movements. If the mere eontraetion of the blood-paths was the ause of convulsions, they ought to eome on at onee, however,

[1] Therap. Mat. Med. and Toxicology, 6th ed.
[2] Ziemssen's Encyclop., art. Epilepsy.
[3] Petersburger Med. Zeitschrift, vol. xi.

and not require prolonged contraction. Again, when a peripheral sensory nerve is galvanized there is not alone a vasomotor spasm of the vessels of the brain, but of the entire body, and as there is no escape for the blood just as much must circulate as before, although under greater pressure. If, however, the blood-vessels were tightened only in the brain, then the theory of Nothnagel might hold good.

The writer believes the convulsions—which were undoubtedly obtained by these observers—to have been due to the nervous irritation produced by the severe peripheral stimulation. Every one knows that if the foot be tightly bound and the sole be tickled with a feather, the sensation is not only soon unbearable, but if persisted results in a typical convulsion. It is a similar condition to that produced by Nothnagel and Krauspe; indeed, the convulsions obtained by these investigators seem to me to support by direct experiment the idea of *reflex epilepsies.*

Langendorf and Zander[1] have produced epileptic spasms by galvanizing the peripheral ends of the vagi nerves, after section, producing thereby cardiac arrest and cerebral anæmia. They found they could do this in chloralized rabbits, but that they failed if the chloral was pushed too far.

We have now adduced most of the evidence that cerebral anæmia produces convulsions, but though all this work has been done it in no way proves that cerebral anæmia is the cause of epilepsy.

In the writer's belief, not only is this method of experimentation exceedingly incorrect, but the principles upon which it was carried on are entirely without support, notwithstanding the fact that some have thought

[1] Centralblatt für klin. Med., No. 4, 1878.

them as indicative of a "convulsive centre" in the medulla oblongata.

Here, as in some of the recent experiments on the heat-centres in fever, the author wishes to insist that the production of absolutely abnormal conditions is not the way to prove the existence of a physiological centre.

One reads that such and such an investigator punctured or removed cerebral areas and found a rise of bodily heat, and so calls the part removed a heat-centre. It is anything but a heat-centre, for if it was what its name implies its removal should produce a fall of body-heat, not a rise. The convulsions of cerebral anæmia are the outward manifestations of the abuse to which the cranial contents are put, and cannot be regarded as scientific measures for the calling out of physiological hebetude or physiological activity.

It having been proved, to the satisfaction of Küssmaul and Tenner, that cerebral anæmia produces convulsions similar to epilepsy, they attempted to discover whether cerebral hyperæmia developed by artificial means so resulted, but obtained nothing but negative results. On the contrary, Landois[1] has supported the theory that hyperæmia is similar in its results with anæmia, while Escher and Hermann[2] have by further experiments confirmed only those of Küssmaul and Tenner. Either some fallacy underlay the work of Landois or else the hyperæmia was so intense as to produce absolute pathological conditions in the brain.

The whole theory that epilepsy is due to cerebral anæmia because epileptiform convulsions occur on its production is a species of reasoning entirely unjustifiable and quite absurd, and is harmful because it blinds

[1] Centralblatt f. die Med. Wissenschaften, 1867.
[2] Pflüger's Archiv, vol. iii.

good investigators so that they seek no further for an explanation.

Some writers have held that the proof of their theory rests in the pallor which comes on in the face at the onset of the convulsion; which, by the bye, it does not always do. These persons forget, however, that, even if this were the case, the disease in reality would exist not in the vasomotor condition of the brain, but in the discharge of a sudden constricting influence from the vasomotor centre. In other words, if their reasoning were carried out logically to the end they would have two morbid changes going on—one primarily in the vasomotor system, the other secondarily in the brain—as its result. The argument, too, that pallor of the face shows cerebral anæmia is foolish, for every time a person blushed cerebral hyperæmia ought to result if this were true.

The writer thinks, therefore, that the idea that we have in cerebral anæmia the immediate cause of epilepsy is absolutely untrue and quite as unlikely.

Quite a number of years ago it occurred to those who were interested in the disease that another and more logical cause was really to be sought after, and that there must be some starting-point in which the explosion of nerve-force originated. As a consequence of this much research has been resorted to to discover whether this area or convulsive spot really existed.

Küssmaul and Tenner (*loc. cit.*), after much experimenting, placed this point in the nervous matter somewhere between the spinal cord and the crura cerebri, or, in other words, in the pons Varolii or the medulla oblongata. Brown-Séquard[1] and Schiff[2] have reached

[1] *Loc. cit.*, p. 130.
[2] Lehrbuch der Physiologie.

similar conclusions. Nothnagel[1] has also promulgated a theory that there is a so-called convulsive centre in the pons Varolii, and that it is from this focus that all the voluntary muscles are involved. In addition to these opinions, we have the conclusion of Hallopeau[2] that such a thing as spinal epilepsy, or epilepsy arising from the spinal cord, may exist, and although no less an authority than Brown-Séquard denies this, and the whole physiological teaching of to-day is against such an opinion, it has been thought best to mention it here.

Even if epilepsy be spinal in origin, the experiments of Nothnagel and others have proved that here, at least, epilepsy is not due to anæmia, for they found that anæmia of the spinal cord is not followed by convulsive movement. Küssmaul and Tenner have also noted this. One must, therefore, either believe in anæmia of the cerebrum as a cause and throw the spinal origin of epilepsy aside, or the reverse, for both views cannot be held together. While the author fears that the reader may regard him ere long as a universal skeptic in regard to the results which such notable men have obtained, he cannot help drawing his attention to some points which are at least worthy of remark, for, after all, it is our duty not to accept blindly any one's results, but to put them to a thorough mental test. According to our present knowledge clonic spasms arise in the cerebrum, while tonic spasms arise in the bulbo-spinal system. So universally is this recognized that the fact that a man is suffering from a tetanic spasm is proof that his spinal cord is affected, either directly or indirectly, provided the contraction is prolonged. Of course, the writer does not mean that no

[1] Virchow's Archiv, xliv.

[2] Das accidents convulsifs dans les maladies de la moelle épiniere. Paris, 1871.

instanee has ever occurred in which an exeeption to this rule has taken plaee, but proof of such an instance is virtually unknown. Neither does he attempt to deny that the investigators named obtained eonvulsions, but he does doubt that the eonvulsive movements were epileptiform in eharacter; at least, as we understand the term today.[1] Thus, if the reader will turn to the literature of these writers he will see that they speak of tonic epilepsy and clonie epilepsy, using the word epilepsy to signify any eonvulsive eondition. Owing to this, it is exceedingly difficult to separate the results really bearing on our subjeet from those which do not. We are, therefore, partly in the dark, so to speak, as to whether clonic movements may be generated in the pons Varolii or medulla oblongata by stimulation; but we know that, generally, if not always, the result is tonic spasm. We have, therefore, no positive proof that true epilepsy finds its origin in these regions, either experimental or pathologieal, and we have greater reasons to doubt this theory than to aeeept it, for Rosenbaeh,[2] working in Mierzejewski's laboratory, has proved that medullary stimulation results not in clonic but tonic spasms, and it seems impossible that a nerve-storm of sueh severity could originate in a portion of the nervous system so pregnant with vital eentres as is the medulla without causing death and general cardiae and respiratory chaos. Knowing the assertions of the others, whom the writer has quoted, Rosenbach has been apparently most eareful in reaehing his results, and denies their eonelusions *in toto.* Further than this, the views of Rosenbach are firmly

[1] It should be remembered that the medulla oblongata is but an enlarged part of the spinal cord somewhat more highly specialized, and although within the skull is really spinal, not cerebral, both anatomically and physiologically.

[2] Vestnik klin. ü Sudek. Psikiatric, vol. ii, fasc. i, p. 171.

supported by the results obtained by Seppilli,[1] and by those of Soltmann and Tarchanow, as well as those of Albertoni. If the medullary theory is true, a skillful hypothesis must be drawn up to explain the loss of consciousness and the fact that the whole body is rarely convulsed all at once, and not by degrees, as is generally the case.[2] Besides all this, we have, on the other hand, much direct and incontrovertible evidence that epilepsy is developed in the cerebrum proper.

Within the last twenty years an entirely new school of experimenters has arisen, led by the celebrated Englishman, Hughlings-Jackson, who may be said to be the father of modern cerebral localization, and this school has done very much more than all their predecessors to decide matters positively.

Probably no one will deny that Ferrier is the chief among this class of experimenters, not only because he was one of the first in the field, but because his results have so brilliantly been supported by his successors in this line. It should not be forgotten, however, that others made some attempt at similar researches before him, as, for example, Nothnagel, or Bright and Todd. Contemporaneously we find his co-workers to have been Vulpian, Charcot, Lépine, and Landouzy, as well as Pitres[3] and Frank, Unverricht[4] and Schroeder von der Kolk. Following these come an equally distinguished band, composed of Luciani,[5] Schäfer, and Horsley.[6]

[1] Rivista Sperimentale di Frenatria, fasc. i and ii, 1881.

[2] Some results reached by Luciani may in the end dissolve the dividing opinions as to the medullary and cortical theories, for he found that extirpation of all the cortex except one leg-centre produced general fits when this centre was galvanized. He therefore concludes that the medulla must act as a distributor.

[3] Trav. du Lab. de M. Marey, 1878-79, p. 413.

[4] Archiv f. Psychiatrie, Bd. XIV.

[5] Riv. Sperimentale de Frenatria, iv, 1878, p. 617.

[6] Proceedings of the Royal Society of London.

We have therefore before us at this point those who have advanced our knowledge in the past and enabled those just named to complete the work which is now being carried out with such ardor, and is blessed with such good results.

As has already been said, the all-important question to answer is, Where does the primary seat of the nerve-storm exist? From one yet undecided the answer might well be: The experiments of Brown-Séquard, of Küssmaul and Tenner, and of Nothnagel point to the medulla oblongata, while those of Ferrier, Luciani, Bartholow, Unverreicht, Munk, Bubnow, and Heidenhain and many others, particularly those of Horsley, point to it as being in the cerebral cortex.

It is impossible for me to detail at length, or even by a shorter method, the labors of the investigators in cerebral localization. The writer can only point out some of the salient parts with the purpose of refreshing the reader's memory who is not constantly reading and studying the accounts of the results as they appear in print.

In the first place, it is a fact known to all physiologists at the present time that stimulation of certain areas in the cerebral cortex produces movements in given portions of the body of greater or less extent, and now science has progressed so far that parts of the cerebral surface can be called by the name of the part they govern; as, for example, the leg-centre, the arm-centre, etc. To speak roughly, the results of all experimenters, when placed side by side, divide the cerebrum into three great areas. The first, or frontal third, in which the processes of thought are probably carried out; the second, or middle third, in which the impulses resulting in movement are generated; and the posterior third, in which the centres

for the perception of sensory impulses reside. The highly developed frontal lobes in the higher races of mankind, and their constant decrease as the degrees of intellect descend, along with the fact that stimulation of these regions produces no appreciable motor effects in the lower animals, are the reasons for these conclusions. The functions of the middle third, or parietal regions, are understood and localized to a most accurate and minute extent, and this at present has gone so far that galvanization of millimetre squares on the cerebral surface is carried out with accurate results. The centres not only for the various movements of a limb have been found, but those which produce certain definite and never-varying motions have been discovered.

A further point of interest has been noted by Horsley, who finds that the centres for various muscles are arranged in such a way as to be divided from one another by horizontal imaginary lines. That is to say, taking the arm-area for example, that the centres for the movements of the muscles of the shoulder are highest, then those for the lower arm and wrist come next, while those governing the hand come lowest. It is generally found that the centres governing large muscles are higher up on the surface of the brain than those governing smaller muscles, and if we stop for one moment to think this seems but natural, since the upper centres are really the masters of the lower ones, or rather the muscles of the shoulder are really the masters of those of the forearm. If we seek to pick up anything we first extend the entire arm and then the wrist.

As regards the occipital area, or the last third, the results have been reached only by negative methods, if one can use such a term; that is, by extirpation and watching the results; for, as it is receptive, not expulsive,

in eharaeter, galvanization produees no external signs of response. This field, by reason of this indirect method of reaehing eonelusions, is not so well mapped out as is desirable, for though one experimenter may attempt to extirpate the same area as his predeeessor the delieaey of nervous protoplasm is so great as to make the slightest elumsy movement felt. This beeomes even more clear when it is reealled that the breadth of a millimetre may involve another funetion entirely.

Still further evidenee has been reeorded by Hitzig,[1] who by the removal of eertain eortical areas governing eertain parts produeed epileptie seizures, beginning spontaneously, either on the next day or in a few weeks. Just here, however, I must call attention to a very important element of fallaey whieh in many ways may have, to a eertain extent, falsified Hitzig's results.

Antiseptics being unknown and healing by first intention being practieally unheard of, it is perfeetly possible and probable that the eonvulsions noted may have been due, at least in part, either to septieæmia or inflammatory eauses.

Munk observed that fits eould be brought about by the exeitation of a limited eortieal area, and eould be stopped by the removal of that area, unless they had lasted for some time. Bubnow and Heidenhain[2] have also shown that in the early part of an attaek extirpation of one eentre quiets the tributary part, while the rest of the body remains eonvulsed. They have also found that in other cases, after rapid destruetion of the motor area on one side, eessation not only on the opposite sides but on both sides takes plaee, it being indifferent whether the area removed was the same as, or

[1] Untersuehungen über das Gehirn, Berlin, 1874, p. 271.
[2] Pflüger's Archiv, xxvi, 137.

opposite to, that by which the outbreak originally occurred. They suppose from this that each area not only governs the opposite muscles, but also that each area influences the opposite area, and that, that failing, the excitation is insufficient to convulse.[1] In cases where the extirpation failed to stop the attack Bubnow and Heidenhain believe that the excitation had reached subcortical areas untouched by the knife. These two observers also made an interesting observation, for they remark that, while fits excited by cortical stimulation always commence on the opposite side of the body, those due to subcortical stimulation begin on the same side of the body. These results have also been confirmed by Unverreicht,[2] who has also proved that section of the corpus callosum does not prevent the nervous impulses from crossing to the other side of the body.

On the other hand, the researches of Rosenbach[3] differ somewhat in their results from those just quoted, for he found that extirpation of a cortical centre does not stop the convulsion in a tributary part. This difference, however, is probably not real, for we remember that Bubnow and Heidenhain[4] found the same thing true if they did not use the knife quickly enough. Rosenbach is, however, entirely in accord with the other observers in finding that cortical stimulation results in convulsions. Ziehen,[5] of Jena, has also determined this, and has found that mechanical irritation of the corpus striatum, of the middle and posterior portions of the thalamus opticus, and of the anterior part of the cor-

[1] Note that these conclusions are virtually identical with those of Dr. Randolph recorded a few pages back.

[2] Arch. f. Psychiatrie, Bd. XIV.

[3] Vestnik klin. i Sudeb. Psikhiatric, vol. ii, part i, p. 171.

[4] Pflüger's Archiv, xxvi, 137.

[5] Versig. d. Südw., Deutsch Neurologen und Psychiatrie.

pora quadrigemina gives rise to energetic movements of locomotion, while irritation of the posterior part of the quadrigeminal bodies causes tetanic convulsions. He thinks that the tonic stage of the epilepsy arises here, but, we think, on very insufficient grounds.

The writer has gone into these results in some detail because they bear most intimately upon what is to follow as part of the evidence which he will bring forward.

When we remember that in nearly all cases of epilepsy due to growths or injury the lesion is found after death, or even during life, to be cortical in situation, we have the finger of pathology to guide us; and we may also state that whenever the tumor is the direct cause of the disease it begins locally, provided the lesion be in the cortex, or, more generally, if it be subcortical.

To one who has experimented and seen the results that the writer has seen in this work, it seems scarcely necessary for him to adduce evidence of the epileptic paroxysm which stimulation of the cerebral cortex may set up. If a moderately strong current be applied to the arm-centre, or any similar point, the limb will respond, and, if the stimulation be continued, will become for the moment tonically contracted, but immediately after passes into clonic movements, which become more and more violent until the rest of the body is convulsed. Not only is the condition exactly similar to the ordinary epileptic attack in every way, but loss of power not only follows temporarily in that limb, but also the centre governing it refuses to respond to further stimulation for the time being.

Can anything be more typical? One has only to see such an experiment to be converted to the belief that epilepsy is cortical.

This is indirectly indorsed by the interesting results

of Albertoni, of Soltmann, and of Tarchanoff, for they found that in newly-born cats, dogs, and rabbits they could not produce fits by stimulating the cortex, for the very evident reason that in these animals the cortex was not sufficiently developed to respond, only the vital centres in the lower areas being developed to carry on life.

Albertoni was able to produce convulsions, however, by galvanizing the cortex of a dog of 23 days.

One of the most interesting proofs of the cortical origin of epilepsy lies in reports of very rare cases by Ocbeke and Gowers,[1] in which a lesion occurring in the internal capsule prevents the appearance of any more nervous disturbances,—at least, on the opposite side of the body from the capsular injury. In other words, the pathway is blocked between the cortex and the lower distributing nervous apparatus.

As has already been said, the character of the aura indicates generally the region of the nervous system which is diseased, and we may use this as a means of localizing lesions during life. Thus, an aura consisting of blindness may point to involvement of the centres governing or connected with vision or situated near by, or, if the aura is olfactory, it indicates cerebral disease in the region of the olfactory bulbs, unless the case be one primarily reflex.

The writer has given, so far, only the physiological evidence of the cortical origin of epilepsy; he will now give the pathological or morbid-anatomy side of the question. First, however, he must, in justice to the subject, give the observations of those who look for the cause of the disease in the medulla and corresponding regions. Indeed, he is forced to include some reports in which every

[1] Nervous Diseases, p. 693.

one has his doubts at the present day in order to present all views to my readers.

In support of the theory of the medullary origin of epilepsy, it is stated that lesions can be found here after death. Thus, von der Kolk [1] found capillary dilatations in the neighborhood of the hypoglossal nucleus in those who bit their tongues during the fit, the dilatations being greater in these than in those who did not bite the tongue by 0.096 millimetre, while in the corpus olivaire it was 0.098 millimetre and in the raphé 0.055 millimetre. When the capillaries of those who did not bite their tongues were examined in the path of the vagus, they were found wider than the others by 0.111 millimetre. The following table illustrates his results :—

Varieties of Epileptics.	Hypo-glossus.	Corpus Olivaire.	Raphé.	Vagus.
(*a*) Tongue-biters, .	0.306	0.315	0.315	0.237
(*b*) Not biters, .	0.210	0.217	0.217	0.348
Difference, . .	+0.096	+0.098*a*	0.055*a*	+0.111*b*

We are prepared to accept all that is reasonable in regard to pathological changes, but can any one explain why tongue-biters' capillaries are here dilated and here contracted, or does any one believe that it is possible to prove that capillaries can be so accurately measured as the 0.111 of a millimetre, and this measurement be taken as a representation of their condition in life when they have submitted to a hardening fluid or, at least, a section-cutter, and have lain dead in dead tissue for hours or days? Such results must be entirely worthless.

Much of the labor of pathologists in the past has reached only contradictory results in so far as the determination of the definite lesions is concerned, unless

[1] Minute Structures of the Spinal Cord, New Syd. Soc. Translations.

the cause be a morbid growth or a lesion from traumatism. Many years ago, Lébert and Dalasiauve [1] noted that selerosis of the hippoeampal folds often existed in epilepties, and their observations have been confirmed in 20 cases by Meynert, Nothnagel, and Chareot, but it is to be remembered that both of the latter believe the changes to be seeondary, not primary, to the epilepsy. Tamburini [2] reports a ease of epilepsy with hemiplegia in whieh there was found induration of the left optie thalamus and the left eornu ammonis, and in which, during life, there was aphasia.

That the presenee of hippoeampal disease is not so common as some would have us to believe seems proved by the results of Hemkes,[3] who found in 27 epileptie males and 7 females, at the Holdesheim Asylum, only 6 with hippoeampal disease. Nothnagel [4] has also injeeted ehromie aeid into the hippoeampi of rabbits without produeing epilepsy, and has also injured this region with punetures without produeing epilepsy. Besides this, Hemkes has removed the hippocampi with no eonvulsive results. When it is remembered, too, that this portion of the brain of man is only rudimentary, while in animals it is much more highly developed, it scarcely seems likely that hippoeampal disease in man would primarily produee epilepsy, as it does not do so in animals.

Pfleger [5] and Hemkes have also found no lesions in epileptie brains save in the eornu ammonis, the ehange being sclerotie in character, and in a later paper Pfleger [6] records 45 autopsies in whieh atrophy of the

[1] Traité de l'Epilepsie.
[2] Sallanzani, Modena, 1879, viii, 550.
[3] Allgem. Zeitschrift für Psychiatrie, Bd. XXXIV, Heft 6.
[4] Virehow's Arehiv, Bd. LVIII.
[5] Allgem. Zeitschrift f. Psychiatrie, Berlin, 1879, xxxvi, p. 359.
[6] *Ibid.*, lxxvi, and Archiv de Neurologie, No. 2, 1880, p. 299.

cornu ammonis with sclerosis was found twenty-five times. He also finds that in every instance the extent of the lesions was in direct proportion to the severity and extent of the paroxysms during life.

In a series of 90 autopsies, Sommer[1] found changes in the cornu ammonis in every instance, and the writer believes this lesion to be present in at least 30 per cent. of all fatal cases of epilepsy.[2]

In many instances, where the disease has existed on one side in a more highly developed form than on the other, and has been associated with a certain amount of hemiatrophy, the autopsy has disclosed corresponding hemiatrophy of the brain. Baume[3] states that the weight of the hemispheres in epileptics is alway unequal, the lightest hemisphere being on the side opposite the convulsed portion of the body. In one series of cases examined by him the smallest difference between the weights of the hemispheres was 15 grammes; the greatest difference was 290 grammes; the mean difference of all the cases being 50 grammes. In another series of 20 cases the smallest difference was 4 grammes and the greatest difference 159 grammes, making the mean 40 grammes. Hamilton[4] has shown that hypertrophy of the epileptogenous side of the brain often exists.

Numbers of cases of epilepsy have also been due to tubercle, and Luys[5] reports an instance in which, after death, the medulla oblongata was found tuberculous. Greenhow, Dresche, Green, and Lobel have also reported a number of such cases.

[1] Archiv f. Psychiatrie und Nervenkrankheiten, Bd. X, Heft 3.

[2] Coulbault (Thèse de Paris, 1884) has written of the presence of this lesion in epilepsy. Those interested I would refer to the original.

[3] Annales Méd. Psychologique, tome viii.

[4] Amer. System Practical Med., p. 491.

[5] Archives gén. de méd., 1869, ii, p. 541 *et seq.*

Imperfect cerebral development has also been insisted upon as a condition commonly found after death, and Echeverria[1] has laid great stress upon the hyperplastic increase in volume of certain cerebral areas. A very interesting study, which brings us face to face with some useful conclusions, has been made by Marie Bra[2] in regard to the general pathological changes seen after death. The results are, perhaps, of sufficient value to be given here:—

1. The mean weight of the brain of epilepties is less than the physiological mean.

2. The cerebellum is greater in weight than the physiological mean.

3. There is frequently an asymmetry between the lobes (not peculiar to epilepsy). The increase in weight is sometimes found on the right side, sometimes on the left. Rarely are both sides equal.

4. In no form of brain disease (except, perhaps, general paresis accompanied by epileptiform crises) have we seen so marked and constant a variation between the weights of the hemispheres as in epilepsy.

In the medulla oblongata Kroon has seen asymmetry also.

Some have believed that the origin of the disease lay in the closure or stenosis of the superior part of the vertebral canal. While the writer has no intention of denying that the observations of all these investigators are correct, they form on collateral information and in reality give us no clue as to the immediate lesions themselves. No one would be insane enough to claim that either cerebral or medullary asymmetry caused epilepsy. These changes form the physiological type, are

[1] Epilepsy and Other Convulsive Disorders.

[2] Quoted by Axenfeld.

entirely secondary to the disease, or have no connection therewith whatever. The writer should also mention the belief of Schroeder von der Kolk,[1] that the medulla is always found to be in a selerotic condition.

It is almost impossible, however, to say this change is primary and this one secondary; we can only call those changes primary where the evidence in regard to them, as being such, is proved. Thus, the changes noted by Greenlees[2] are evidently secondary, for he found that they resembled those of prolonged cerebral congestion. The muscular coat of the blood-vessels were thickened, and the large ganglion-cells atrophied.

With a very few more remarks as to the morbid anatomy of epilepsy, the writer will pass on to the question of the changes seen in epilepsy due to syphilis, embolism, and similar conditions. Within the last two years a very interesting paper has been published by Dercum,[3] on the brains of twelve epilepties, and the author does not hesitate to quote his studies here. In all of these brains abnormalities of the sulci and gyri were found, and in several thickening of the skull was also present. In some of the brains there was overdevelopment of the occipital lobe, with abnormal sulci. In several others, the parieto-occipital and interparietal sulci were confluent, producing an arrangement similar to that of monkeys. In a number of cases the fissure of Rolando opened into the Sylvian fissure. Abnormal sulci were also found in the frontal lobe. In two of the brains the cuneus and in another the lobus quadratus were enormous. In one case an entirely abnormal sulcus traversed the first temporal gyrus. While in nearly every

[1] *Loc. cit.*

[2] Journ. Ment. Science, October, 1885, p. 353.

[3] Proceedings of the Philadelphia Neurological Society, Dec. 26, 1886.

one of the 12 cases there was evidence of mechanical hindrance to brain development, there were also pathological changes going on.

Zohrab[1] has recently published the records of an examination of several brains of epileptics, in all of which he found necrosed and softened spots around and beneath the horns of the lateral ventricles.

In syphilitic epilepsy the lesions producing the trouble are much more understood than those of the idiopathic type, and a very large amount of literature has been written concerning them. In a series of 21 cases of this disease, Echeverria[2] found general scattered lesions all through the brain in 8 cases, in the motor zone in 4 cases, in the temporo-sphenoidal in 2 cases, and in the occipital area in 3 cases. In 4 other cases there was disease of the base of the skull. In 10 instances there was atheroma of the cerebral arteries, twice there was aneurism of the Sylvian artery, and once of the basilar artery. In addition to these changes, there were gummata in the cortex, or the substance of the hemispheres and ganglionic centres, or there was cerebral sclerosis. These changes do not, however, always occur even in syphilis, for not only has clinical experience shown me many cases of the disease where no changes could be detected, but also Charcot and Pitres[3] have recorded the most typical Jacksonian epilepsy without any demonstrable lesions whatever.

That gummata are quite capable of originating convulsive seizures by their presence is proved by daily experience. They may act by so interfering with the

[1] Archives de Neurologie, May, 1886.

[2] Journ. Ment. Science, July, 1880, p. 165.

[3] Nouvelle Contribution à l'étude des Localisations motrices dans l'Écorce des Hemispheres du Cerveau. Revue Mensuelle de méd. et de Chir. Nov., 1879, p. 814.

nutrition of cells as to render them diseased all about that region, or by the irritation which they produce by their presence.

Thrombi and emboli are also the primary causes of convulsions, both in syphilis, in rheumatism, and in cardiac disease, as well as in many other somewhat similar conditions. The rule is that a tumor of the brain or an embolism does not produce convulsions because it is a tumor or an embolism, but because it is situated in an area pregnant with the possibilities of convulsion. Abscess acts in the same manner, and depressions of the skull from injury or effusions of blood may so result.

The pathology of epileptiform migraine is, perhaps, one of the most readily explained points in the discussion of epileptiform disease. It will be remembered that Jackson and others have considered that all cases of true migraine are really evidences of disturbances in the sensory portion of the cortex in the same manner as epilepsy may be due to irritation of the motor portion. In the first place, the very character of typical migraine is epileptic, for it is generally preceded by hemianopsia for a varying length of time. Spasm of localized muscles near the area of pain is not rare, and spasm of muscles and centres elsewhere may be present, as in the case quoted when speaking of the symptoms.

It has been held, too, that the vasomotor and pupillary changes so often seen in migraine are due to the extension of irritation into the medulla oblongata and the cilio-spinal region of the cord.

The pathology of plumbic epilepsy is much more simple than that of the idiopathic group, for we find that there is always some more or less well-marked change in the brain, consisting in a fatty degeneration

of the large cells, and afterward some atrophy. The blood-vessels in this state are generally atheromatous, or at least thiekened, and the lymph-spaees show evidences of wide-spread ehange. In some instanees the post-mortem examination reveals very aeute and very severe inflammatory ehanges, such as acute cerebritis, but in others a more subaeute or ehronie ehange only seems to be present. The general system elsewhere is often found even more profoundly affeeted than the brain, and the kidneys are almost invariably diseased more or less. Indeed, so eommon is it to find renal disorder in these eases that some have attempted to prove that the epilepsy of lead was really uræmie. I have shown under the head of diagnosis that this is a mistake, and that they ean generally be separated. We have, to be sure, an epileptiform attaek due to uræmia produeed seeondarily by lead, but we also have one in whieh the lead acts direetly. Rosenstein[1] has poisoned dogs with lead in sueh a way that ehronie poisoning resulted, and has seen epileptie fits produeed in these animals in this manner; but he states positively that they were not uræmie in eharaeter. He also found in these eases that the lead eould be reeovered from the brain in large quantities, and this is, as is well known, in aeeord with hundreds of other observers who have proved that, in ehronie lead-poisoning, the metal is to be found in every tissue of the body, even to the eoats of the blood-vessels.

What has already been said must have enabled the reader of this essay to see that epilepsy is essentially a eortieal disease of the eerebrum, brought about, of eourse, in many ways. Either direet irritation of eertain cells may so result, or indireetly by irritation of a reflex eharaeter they are perverted from their normal

[1] Virchow's Archiv, 1867.

function. No one, be he ever so wise, will be able to tell the reader the ultimate cause of the nervous discharge which causes the attack, until some one has discovered the manner in which the remote something which causes nervous protoplasm to give forth impulses acts. Above all, the author desires to impress the idea that epilepsy does not signify a disease, but a symptom of a disease. It should not, strictly speaking, be employed or applied respecting the condition which is now indicated by it any more than the word dropsy should be used to indicate nephritis. Epilepsy is the manifestation of morbid nervous changes, even as dropsy is of renal or cardiac lesions.

Diagnosis.—When speaking of the symptoms of epilepsy the writer has so thoroughly described them in all their details that, under this heading, he will rather devote his efforts to the question of differential diagnosis.

Undoubtedly, the most similar convulsive condition that we have is that known as hysteria, and the diagnosis of one from the other is as difficult in some cases as it is essential and necessary for treatment and cure. The other conditions, with which it might be confused, are uræmia, alcoholic epilepsy, tetanus, and syncope. On the following page are arranged all these disorders in a table, which briefly and succinctly shows the different points between them, although, of necessity, it is somewhat arbitrary on account of the lack of space. Nevertheless, it is hoped that it will be clear enough to be of service, particularly in connection with what the author is about to say.

The very irregularity of true epilepsy makes it extremely difficult to give clear and well-defined outlines of it against another disease, particularly when we

TABLE OF DIFFERENTIAL DIAGNOSIS OF EPILEPSY FROM HYSTERIA, ETC.

Signs.	Epilepsy.	Hysteria.	Uræmia.	Petit Mal.	Alcoholic Epilepsy.	Tetanus.	Syncope.
Apparent cause.	None.	Emotion.	None.	None.	None.	None.	Mental shock.
Aura or prodromata.	Generally present but short.	Globus hystericus. Palpitation. Choking.	Headache, vomiting, and dyspepsia.	Faintness and dimness of vision.	Tremors.	Nervousness.	Not so well defined as in epilepsy.
Onset.	Sudden.	Often gradual.	Often gradual.	Sudden.	Sudden or gradual.	Gradual. Begins in jaw.	Sudden or gradual.
Scream.	At onset and sudden.	During attack.	Frequently none.	Frequently none.	May or may not be present.	None.	None.
Convulsion.	First tonic, then clonic.	Rigidity more pronounced, with more aching.	Rigidity generally absent.	No rigidity.	Movements more clonic than tonic.	Always tonic.	None.
Biting.	Tongue.	People, tongue, lips, and hands.	Tongue.	None.	Rarely.	None.	None.
Micturition.	Frequent.	Never.	Never.	Rarely, except when bladder is affected.	Rarely.	Sometimes.	Never.
Defecation.	Occasionally.	Never.	Never.	Never.	Rarely.	Rarely.	Never.
Talking.	Never.	Frequent.	Muttering.	Never.	Never.	Never.	None.
Duration.	A few minutes.	Generally many minutes.	From a minute to hours.	Momentary.	May be prolonged.	Hours.	Indefinite time.
Consciousness.	Lost.	Generally preserved.	Lost.	Not lost always, but clouded.	Lost.	Preserved.	Lost.
Termination.	Spontaneous.	May be induced by shock.	Spontaneous.	Spontaneous.	Spontaneous.	Spontaneous.	Gradual, with no somnolence.

remember that epilepsy and hysteria often go hand in hand.

By far the most important differential point between the two disorders just named, when not complicated with still another disease, is the character of the movements. As already pointed out, in epilepsy they are typically at variance with those of daily life, while in hysteria they are almost equally typical of ordinary muscular contractions, or, in other words, are more purposive in character; and frequently there is prolonged tonic contraction of the muscles, giving rise to the assumption of positions which bear more or less resemblance to normal attitudes. In hysteria, also, consciousness is impaired sometimes, but never so completely as in true epilepsy. Indeed, most commonly the individual knows all that goes on around her, for, while she may give no sign of consciousness by words or looks during the attack, she may afterward be able to narrate all that has occurred. Less commonly, however, a condition known as automatic consciousness exists, in which, during the paroxysm, the patient understands all that is said, but forgets everything on the return to quietness.

The fact that the patient is a female cannot be regarded as affirmative evidence of hysteria in the least, but the condition occurring in a male may be taken as fairly positive evidence of its being epilepsy; and yet it should always be remembered that males may suffer from hysteroid attacks.

The movements of the hysterical patient after the tonic condition has passed away are as clonic as those of epilepsy, but still possess some purposive characteristics, and are not so *bizarre* as are those of the true disease. Thus, the head, arms, and legs, are struck with evident endeavor against the floor or surrounding

furniture. Another point, which, when it occurs, is very distinctive, is the onset, toward the close of a hysterical convulsion, of a second stage of tonic spasm such as occurred at the beginning. It will be remembered that this does not occur in epilepsy, although it must be borne in mind that in cases of the "status epilepticus" the rapid onset of another attack may show a second tonic stage. This can be separated, however, by the fact that it is followed by clonic movements, whereas the secondary tonic stage of hysteria is usually followed by relaxation and temporary recovery.

In the secondary hysterical tonic contractions emprosthotonos and opisthotonos may occur, and are even more rigid in their character than they are in the first, in some cases. Finally, too, in hysteria, some peculiar emotional position is often assumed, as of the crucifix, or of intense grief, or, perhaps, immoderate laughter, with corresponding movements of the trunk. If the patient is quiet at this time, a smile may float across the face, while the eyes, with a look of pleasure, pain, or entreaty, may seem to be gazing at some object very far off. In some very well developed cases the expression of pleasure is followed by a look of pain, with painful movements, or an intense appearance of voluptuous entreaty, with sensual and venereal desire evidenced by gestures. Great terror may be present, and, as the scene constantly changes, the woman is now joyous, now mournful, now scolding, now praising her attendants or herself. Such is the history of a fully-developed attack of hysteria.

Hysteria is rarely seen among the Germans, Belgians, or corresponding races, but very frequently observed by French practitioners of medicine.

In France there can be no doubt that the tongue is commonly bitten in hysterical convulsions, and that

frothing of the mouth is frequently present; but in the other countries which have been spoken of its presence may be regarded as indicative of epilepsy rather than hysteria. Doubtless the inexperienced reader will say, upon comparing these symptoms with those which were given as occurring in epilepsy proper, that the two disorders are easily separated from one another; but the author must insist most strenuously upon the fact that in both cases he has only given the most typical characteristics of the diseases, and he repeats that all cases are not by any means so well defined. He would also remind the reader that the chief difficulty in making a diagnosis lies in the fact that frequently it must be found without any previous history of the case, as when a patient is brought into a hospital, in a fit, for treatment. Where the history is obtainable, or where the diagnosis can be put off until the case may be studied, the question becomes more simple.

If a large number of patients suffering from these hysterical attacks be questioned in between times, it will be found that the so-called *globus hystericus* becomes an almost constant precursory symptom of an attack, and if the relatives be questioned it will often appear that they have noticed that the fall to the floor is more gentle than in true epilepsy; but this is not always so, by any means. Again, the expression of the face in hysteria is, between the attacks, often very characteristic, and the surrounding atmosphere of the patient seems, even to the inexperienced, to breathe hysteria. Very commonly areas of anæsthesia and hyperæsthesia occur in these patients, and are of all degrees of intensity and limitation. Search for them generally shows their presence after attacks of convulsions, but they may exist from one attack to the other, or develop spontaneously. In

nearly all cases these areas are unilateral, and may extend entirely over one-half of the body, the line of demarcation of the anæsthesia or hyperæsthesia, from the sound area, being clearly and abruptly defined, generally at the median line of the front and back of the trunk. It will be called to mind that such conditions are very rare in true epilepsy. Hallucinations are far more common after the fit in hysteria than in epilepsy, and sometimes they even occur during the attacks. They are always associated with the mental states; in terror, rats or disgusting objects are seen, and, according to Charcot,[1] are generally seen on the side which, during the intermissions, is anæsthetic. The pupil is more mobile in hysteria than in epilepsy, but may be contracted, normal, or widely dilated.

The following table gives, in as brief a manner as possible, the differential diagnosis between epilepsy and hystero-epilepsy, and is founded on a lecture by Professor Charcot,[2] delivered at the Salpêtrière. Aside from its conciseness the writer has inserted it here because of the standing of its partial originator, whose opinions on such subjects are, of course, of immense value by reason of his ability and opportunities :—

True Epilepsy.	Hystero-Epilepsy.
Aura short.	Aura extremely prolonged.
Cry is violent.	Cry is more moderate and prolonged.
Spasms first tonic, then clonic, then followed by stertor.	Ataxic contractions, extension of limbs, turning of head, clonic movements, slight stertor.
Sometimes after fit delirium or violent impulse or mania.	*Bizarre* contractions, no delirium, may be hallucinations.
Mental power is lost.	Mental power preserved.
No emotional attitudes.	Emotional attitudes.

[1] Leçon sur les maladies du système nerveux. Paris.
[2] Gazette des Hôpitaux, 1878.

A very useful differential point, strongly insisted upon by Charcot and Bourneville, is that in true epilepsy there is generally a very considerable rise of temperature during an attack, while in hystero-epilepsy the temperature remains normal or only slightly raised.

Some observations, made by Charcot[1] in "status epilepticus," showed that the temperature in this condition rises to 40° or 41° C. (104° or 106° F.), or even as high as 42° C. (107.6° F.). Under these circumstances death often comes on rapidly. In hystero-epilepsy, on the other hand, Charcot found that the bodily temperature always remains at 37.5° to 38° C., and death is exceedingly rare or never occurs. These observations have been found true by many other observers, and there can be no doubt of their truth.

Some authors state that a continuous rise of temperature in hystero-epilepsy is sufficient to establish the presence of some other disease, or else a very threatening condition of the nerve-centres. Thus, in a case reported by Quincke,[2] after a series of apyretic convulsions, convulsions occurred with elevation of the temperature to 43° C., and soon ended in death.

In the diagnosis of true epilepsy from convulsions of a hysteroid character, it is well for the physician to remember that the proportion of the two conditions in frequency of occurrence is, according to Gowers (*loc. cit.*), 815 to 185 in every 1000 cases.

The differentiation of epilepsy from uræmia is much more readily carried out, for there is usually a previous history of symptoms pointing to renal trouble, as, for example, some œdema, or somnolence, or mental apathy, for some days or hours before the attack. Of course,

[1] Le Progrès Médical, Jan. 10, 1874.
[2] Archiv f. Heilkunde, 1864.

in such cases, recourse may be had to the ordinary tests for such conditions of the urinary organs as are generally found where uræmia exists; but it is to be remembered that epilepsy and kidney disease may exist hand in hand, and that, for this reason, the prognosis and diagnosis are to be carefully formed and given. If, in a given case, a prolonged history of dyspepsia, of frequent vomiting, occasional attacks of asthma and failure of general health is found to be present, the diagnosis ought probably to be uræmia. The preservation or loss of consciousness in uræmic convulsions is variable. Generally if the convulsion is wide-spread and severe the intellection is lost, but if it be only a slight attack it may be preserved. So long ago as 1840 Dr. Bright described cases of uræmia, on the other hand, in which furious convulsions occurred without loss of consciousness, and Roberts has reported similar instances.

Just here the author may remind the reader that not more than thirty years ago some physicians of very high standing believed epilepsy to be due entirely to uræmia. Thus, Sieveking[1] firmly believed in this theory and reported a case in support of his views. Fatal uræmia may also occur in a patient whose urine is apparently normal; and, in a large number of cases of chronic contracted kidney, albumen may be absent from the urine for long periods of time. The specific gravity of the urine should be carefully noted, and in very doubtful cases careful estimations of the urea be made. If the specific gravity is constantly below 1.010 the kidney will nearly always be found contracted unless diabetes exists. Tests of the urine passed at different times of the day should always be made. Another means of testing the integrity of the kidney is to administer

[1] Brit. Med. Jour., March 20, 1858 p. 235.

iodide of potassium and study its elimination. It is affirmed that, after a full dose, this drug can in an hour be readily recognized in the urine by adding nitric acid and then starch; but when contracted kidney exists the iodide fails to appear or is excreted only in very small quantities. The temperature of the body may also be used to differentiate between uræmia and epilepsy. In 1865 Kien[1] called attention to the fact that even when uræmic convulsions are most violent they are accompanied by a fall of temperature of as marked a character as the rise noted in respect to epilepsy. Since then, this has been confirmed by Roberts, Hirtz, Hutchinson, Charcot, Bourneville,[2] and Teinurier.

The diagnosis between puerperal eclampsia and epilepsy consists chiefly in the acuteness of the attack and the fact that with no previous convulsive history a woman becomes suddenly convulsed during the puerperal state. This is not a place for the discussion of the identity of uræmia and puerperal eclampsia, although we believe that this is generally supposed to cause the nervous disturbance. If the convulsions are uræmic, the temperature, according to the investigators just quoted, should fall, but according to Bourneville (*loc. cit.*) they are distinctly separated from those of uræmia, by reason of the fact that the temperature rises with great rapidity in the very beginning of the convulsions, and there remains with great steadiness. The condition of bodily temperature cannot, therefore, be used to differentiate puerperal eclampsia and epilepsy.

It is important to determine whether idiopathic epilepsy can be separated from that due to syphilis

[1] Gazette Méd. de Strasbourg, 1865, p. 12.

[2] Études clin. et thera. sur les maladies du système nerveux. Paris, 1873.

simply by the symptoms. Of course, this is very difficult to decide; but the answer to a question of this character ought to be that, so far as the convulsion itself is concerned, it is not possible to separate them. If, however, we can obtain any history the matter becomes much more simple. It is characteristic of syphilis to have severe darting or aching pains in the tibiæ, particularly at night, and it is also characteristic of syphilitic epilepsy to have severe frontal headache before the attack, while in idiopathic epilepsy this pain generally follows the seizure.

Fournier, in his lectures on epilepsy, in the Louvain, in Paris, in 1875, gave a summary of his views as follows:—

1. In syphilitic epilepsy there is nearly always absence of the shrill cry at the onset, so characteristic of the idiopathic variety.

2. There is frequently paralysis immediately after the attacks.

3. The seizure is incomplete or unilateral in character.

4. Attacks constantly increase in severity.

A therapeutic point, which may be used with the greatest success, is the administration of iodide of potassium in large doses. If the epilepsy be syphilitic, it will rapidly become less severe, and enormous amounts of the drug will be borne with impunity. As much as 30 grammes will often do no harm in twenty-four hours.[1]

It has been thought by some that the mental hebetude, between the attacks, is greater in syphilitics than in others. This depends very largely on the area of the cerebrum involved, and not upon the disease itself.

[1] I find on reading several papers on the subject that Fournier has also great confidence in this test.

Of course, if there is a history of a chancre, or any syphilitic scars or erosions are to be seen, the diagnosis is manifest. It is very common in syphilitic epilepsy to find that the attacks are followed by prolonged attacks of paralysis, not due so much to the exhaustion of the centres as to the irritation produced by the gummata or the inflammation which sometimes springs up around them. It is also a noteworthy fact that the paralysis most commonly seen involves the motor ocularis, abducens, and patheticus.

The diagnosis of syphilitic epilepsy from the idiopathic form is of the utmost importance, since the ultimate result must be largely governed by the cause. Dowse[1] has analyzed no less than 274 cases in order to discover any useful points in this respect. He insists, as the writer has already done, that epileptic attacks beginning after 30 years of age are almost surely syphilitic, particularly if no history of traumatism or heredity is present. It is also found that, if some degree of mental alienation is present between the paroxysms it will generally yield to specific remedies. Cyanosis is less frequent and pallor is more common than in the ordinary disease.

It is unnecessary for me to state once more that petit mal is but a variety or modification of haut mal. Nevertheless, it is useful to be able to separate it somewhat from the more severe form of the disease in the attempt to form a prognosis.

Some suppose that petit mal may be designated as consisting of one or two of the chief symptoms of epilepsy proper, and others have thought that the preservation of consciousness was the chief dividing-line. The last idea is certainly incorrect; but it is impossible

[1] Practitioner, Oct., 1878.

to give any outline which will absolutely separate the two conditions, so far as symptoms go. An important and useful point first discovered by the celebrated neurologist, Weir Mitchell,[1] is that, whereas the inhalation of amyl nitrite stops true epilepsy, the use of this drug increases the severity of an attack of petit mal.

Alcoholic epilepsy occurring during an attack of *mania â potu* is, of course, easily diagnosed, and the general appearance of the patient, combined with his history, suffices to decide the physician. The movements are more clonic than tonic, and often are lacking in force. There is, however, no constant distinction between the symptoms applicable to all cases. Generally one seizure of alcoholic epilepsy follows the other every few minutes until three or four have taken place, when the paroxysms cease. It is not to be forgotten that alcohol may produce all degrees of epilepsy, from the mildest petit mal to the most severe paroxysms; and it is also to be remembered that hallucinations of terror are very commonly present.

There may be an aura in alcoholic epilepsy quite as marked as in the true disease.

The separation of syncope from epilepsy is one of the easier tasks imposed upon us. The color of the face, the weakened heart-beat, sudden loss of consciousness, and the general appearance aid us here very much.

The separation of epilepsy from hemicrania has been very well written of by Silva.[2] He thinks that epilepsy begins in childhood below puberty, most commonly, while hemicrania comes on after puberty; and that the attacks of hemicrania decrease in violence and frequency as age increases, while the contrary rule applies to

[1] Philadelphia Med. Times, vol. v, p. 553.

[2] Giornale di Neuropatologie, fasc. i and ii, 1885.

epilepsy. It will be seen that these views are in accord with those of Strümpel and Wagner (*loc. cit.*).

Before closing this section of the essay the writer must bring forward the points to be used in differentiating epilepsy from those attacks simulated by malingerers. Often this is most difficult; and it is related of Fournier that, after his expressing an opinion that a man could always tell them apart, one of his assistants threw himself to the floor on his next visit, in a pretended attack, whereupon Fournier, completely misled, exclaimed, "Poor M. ——, he is epileptic," upon which the assistant, smiling, arose to his feet and confuted the statement.

Very serious injuries are sometimes submitted to by these persons to carry out their designs. Thus, the famous case of a man named Clegg may be cited, who, to deceive a suspicious physician, threw himself, in a pretended fit, to the iron floor of a jail, the distance being 20 feet. The points to be looked into are: the condition of the pupils, which, in the simulated attack, always react normally, nor can the corneal reflexes be held back, the color of the face is rarely changed, and the thumbs are rarely flexed as they should be. Marc[1] has pointed out that in malingerers the by-stander can readily straighten the thumbs out, and that they remain so; whereas, in epilepsy they instantly become flexed again.

Suggestions as to movements are sometimes followed by malingerers, and the movements generally lack the *bizarre* character so typical of epilepsy.

If tobacco or ammonia be held to the nose of the fraud, he generally is forced to disclose his true nature.

The fact that in malingerers there is no rise of temperature may also serve as a differential point.

The diagnosis of lead epilepsy from the idiopathic

[1] Dict. des Sciences Méd., vol. xii, p. 542.

varieties is somewhat difficult, if the patient is seen for the first time during an attack, but the ordinary methods of determining chronic lead poisoning are, of course, of equal value here. The blue line on the gums may be present, and, if so, the diagnosis is almost certainly lead; but its absence is no proof that lead is not present. The administration of iodide of potassium also will so increase the elimination of the poison as to benefit the case and render it more easy to recover lead from the urine.

The history of exposure to lead in any form is, of course, exceedingly valuable evidence, but it should not be forgotten that in many cases this history is wanting. Thus, the poison may be due to a hair-dye, or cosmetic, or to water which contains lead from pipes, or an endless line of similar hidden and obscure causes. Amaurosis may be present in some cases, or optic neuritis with atrophy may occur. Where double wrist-drop is present the diagnosis may be much more easy.

It is exceedingly important to differentiate between those convulsions which arise from the uræmia brought on secondarily by an action of the lead on the kidneys and those which are due to a direct action on the brain. This may be difficult from the mere symptoms presented, but there are some points of difference. In the first place, the convulsion of uræmia is, as a general rule, not so violent in its movements, nor so sudden in its onset. It is generally preceded by a few days of somnolence, or weeks of gastric disorder and headache, while lead epilepsy is generally sudden, or preceded by cephalalgia by only a few days or hours. Again, examination of the urine in uræmic convulsions will show a decreased amount of urates in proportion to the quantity of urine passed, while in plumbic epilepsy just the reverse will

be true, unless the kidneys are affected *pari passu* with the cerebrum. If albumen be present, uræmia is pointed to; but if the urine has a low specific gravity, and is passed in large amounts, the indications are that there is chronic contracted kidney, which may or may not be the cause of the nervous disturbance. (See diagnosis of uræmia, several pages back.)

Aside from the symptoms of epilepsy which have been given, one or two additional facts may be worthy of record. It has been claimed by Addison[1] and others that epileptics have certain physiognomical characteristics, particularly if insanity also be present. Addison, in 50 cases, made up of 39 males and 11 females, recorded these signs as follows:—

	Males.	Females.	Total.
Face pallid,	24	6	30
Lips thick,	20	7	27
Eyelids puffy,	33	9	41
Pupils large,	20	5	25
" medium,	19	6	25
" unequal,	0	2	2

The general aspect presented in chronic epileptics is certainly as these figures represent it to be; but it has already been described so thoroughly that the writer will not do so again.

If there is a history of spasms in a case coming to our knowledge, in which we find asymmetry of the head and face, scarred tongue, and thick, puffy, sensual, or brutish lips, there should be very well founded suspicions that the man is suffering from epilepsy. The skin in such cases is cold and clammy, particularly about the hands, and lacks its normal tone and color. Besides this, it in many cases gives rise to a peculiar musty odor.

It would be out of place for the writer, at this point,

[1] Journal Ment. Sci., vol. xii, 1867.

to consider the surgical diagnosis necessary before the operation can be performed to relieve an epilepsy dependent on a tumor, an abscess, or any similar condition. Both the question as to the character of the lesion and its seat must be settled, and not only a minute study of cerebral pathology would be necessary for a complete mastery of the matter in hand, but also a thorough understanding of cerebral localization, which it is not in the writer's power to give in an essay on epilepsy.

As an illustration of the value of cerebral localization in diagnosis, let the author just here give a case shown to him by Dr. Hughlings-Jackson during his visit to London.

A man, aged 20 or 25 years, with angular curvature of the spine, began to have slight evidences of the so-called Jacksonian epilepsy in the muscles governing the thumb of the left hand. These attacks increased in force and frequency, and gradually involved the entire body. At Dr. Jackson's request, Mr. Horsley cut down over the region known as the thumb-centre, and found a small tumor pressing on the surrounding parts. This he removed, the wound healed by first intention, and the man has had but one or two mild attacks since, and these soon after he was operated on. The left thumb is now paralyzed, but the epilepsy has ceased. It may also be stated, in order to show what unfavorable cases recover, that the spinal curvature is sufficient to produce a paraplegia, which still remains.

Beevor[1] has attempted to show that there is a relationship between the giddiness which sometimes constitutes the aura of epilepsy and the direction in which the patient primarily rotates, using this as a means of diag-

[1] Brain, Jan., 1884.

nosis in those cases where no history can be obtained as to the primary movements from the patient's friends; that is, he finds that the patient can generally recall the direction of the giddy sensation felt beforehand, while the friends may have overlooked the following movements. The value of the point, supposing it to be true, rests upon the localization of the side of the brain most affected. In 17 cases examined by him all of them felt giddy in the direction in which they ultimately moved, or, in other words, the surrounding objects passed in a stream toward the side to which the head was about to be turned. In all these cases he was careful to distinguish between simple giddiness and that due to ear disease or faintness, using only the purer cases, where no manifest lesion was apparent.

Prognosis.—The physician can always assure the patient and friends that so far as the disease is itself concerned there is little danger of death, since, as a general rule, unless the attacks are very severe, death rarely occurs, unless indirectly, by the fall of the body into a stream, or well, or when in some position where a steady head is necessary for safety. Accidental asphyxia, due to the burying of the face in the pillow at night, or to the impaction of food in the larynx, may occur, but even this accident is uncommon. Some superintendents of insane asylums in which epilepties are cared for have resorted to shields, which, being worn over the face at night, holds the mouth so far away from the pillow as to prevent the supply of air being shut off. As the tendency to turn on the face is rarely seen, this danger is overestimated.

The question which the friends will always ask is, What is the prospect of ultimate recovery, or, at the

least, will there be any progress toward an improvement? Unfortunately, the reply ought not, in any case of the idiopathic form, to be favorable, even for ultimate improvement, for the experience in the past of every large practitioner has been that cures rarely occur. Several points which have a favorable bearing may, however, be offered in consolation, as lightening the severity of the sentence; for it will be remembered that, in many instances, if the disease is taken powerfully in hand early in its life and in the life of the patient, the results are certainly fairly good. This is particularly true if the disease seems to be mild at the beginning. Too much encouragement should not be held out from the use of drugs; but this should not be impressed upon the patient's mind, since it is sure to render him careless in taking the remedies prescribed. The influence which sex exerts on prognosis is doubtful, although one or two recent writers (Gowers, for example) think that it is slightly better in males than in females. If this is true, the reason of it may lie in the greater strain placed upon females at the age of puberty.

Curiously enough, hereditary predisposition does not seem to increase the gravity of the prognosis, but rather to improve it, for Herpin[1] and Gowers[2] have both found this true. The latter attempts to explain this by the hypothesis that, as the primary tendency is present, it requires only a slight cause to excite it, which cause is so slight that it is readily overcome by treatment. The knowledge of the frequency of attack is also very important to the physician in forming a prognosis, as is seen by the following table made by Gowers. It is certainly sufficiently convincing in its figures, and for

[1] Du pronostic et du traitement curatif de l'epilepsie, p. 515. Paris, 1852.
[2] Epilepsy, p. 246. London.

this reason the writer takes the liberty of inserting it here.

In 100 cases the results were as follow :—

	Cases.		Percentage.	
	Unimproved.	Arrested.	Unimproved.	Arrested.
Attacks daily,	7	1	18	1.8
Daily or weekly,	11	29	29	46.7
Eight days to 1 month,	16	15	42	24.2
Over 1 month,	4	17	11	27.3
	38	62	100	100.0

It has also been claimed that the presence of an aura not only improves the prognosis by reason of its enabling the patient to get out of harm's way, but also seems to be associated with more remediable forms of the disease.

These points are to be used in making predictions for the future in *idiopathic epilepsy only.* They are in no way of value in other epilepsies, as will be pointed out, but other things take their place.

In the first place, the very fact that idiopathic epilepsy arises without a cause makes it unfavorable, since we know not with what we have to deal; whereas in reflex epilepsy, or that dependent upon tumor, abscess, or depressed bone, the ultimate result depends very largely upon collateral facts, such as the situation of the lesion, the safety of its removal by operation, or the possibility of its removal by drugs, as in syphilis. In regard to syphilitic epilepsy, it may be asserted, with no fear of contradiction, that it can, in the majority of cases, be cured, and in nearly all cases improved. We find that idiopathic epilepsy has, therefore, the most gloomy prognosis, while syphilitic epilepsy has the most promising.

The writer must issue a word of warning, however,

which often by its absence leads to disappointing results, namely, very frequently epilepties, be the cause of the disease what it may, improve greatly under proper treatment for a short time, and then go no farther, or perhaps move very slowly. The primary rapid improvement deceives the physician and friends, whose encouraged thoughts should be held in check, lest they be ultimately disappointed.

In post-hemiplegic epilepsy the prognosis is not favorably for obvious reasons, because the lesion produced is one which drugs can only remedy very slightly, and in which operative procedures are futile.

(For some of the other conditions influencing the prognosis of epilepsy, see the section on Complications.)

Treatment.—After all, the treatment of epilepsy is, to say the least, one of the most important questions which come before the physician, and the only value of all our pathological knowledge lies in the aid which it brings us in combating the disease. Unfortunately, scientific physicians are too frequently inclined to study morbid processes as far as they are interesting, neglecting to use their store of facts for the good of future sufferers, by attempting to argue out of them sensible therapeutic measures founded on a scientific basis and not on empiricism.

The author will not follow out in this essay the common custom of detailing remedies as useful or not useful, and of recording cases where each remedy has produced a cure; but will endeavor not only to name the drugs from which relief is obtained, but also explain why they do good, whenever this is possible, in the light of our present physiological and pathological knowledge. It will be evident, from what follows, that the treatment of epilepsy in the past has been as unwise as the treat-

ment of every other disease, and the remedies have been given in one case solely because they acted happily in its predecessor, who had the same manifestations of the disorder. It is just this senseless form of medication which breeds the contradictory reports of the medical press regarding certain remedies.

It should also be borne in mind that the treatment of epilepsy is as various as the disease is variable in its forms and phases, and should, in nearly all cases, resolve itself into two or perhaps three divisions, consisting in the removal of any exciting cause, in the checking of the convulsive tendency already set up, and in the prevention of any further attacks by suitable drugs or other measures of relief.

The treatment is governed largely by the cause, and is medicinal or operative, according to the etiological factors at work. In the simple idiopathic epilepsy medicinal means must be followed, while in a case resulting from traumatism the depressed bone, abscess, or tumor must be removed. In those due to reflex irritation the peripheral source of trouble must be sought out and relieved.

The writer will first consider the use of drugs, merely prefacing what he says by remarking that, in some instances, medicines and operative measures must go hand in hand.

By far the most useful drug in use to-day for the relief of epilepsy is bromide of potassium, although other forms of bromide salts are to be mentioned later on. It is useless for the author to attempt to give statistics as to the truth of his statement, for every voice in the profession supports it; and the fact has become so generally recognized that very few papers, comparatively speaking, appear concerning it in the medical journals of the

present day. This drug is not, however, a "cure-all," even in epilepsy, and reports are constantly made of cases where it has failed; but in many cases the remedy is undoubtedly responsible for a cure, when it is pushed in a suitable manner, and, in the vast majority of instances, the seizures are so decreased both in violence and frequency that its use may be said to be indicated in every case of the disease. In a very small minority, however, it signally fails, and in a still smaller number of cases it is useless unless combined with some other drug whose power alone is very slight. Nevertheless, it is to be laid down as a rule that the bromide treatment of epilepsy is, *par excellence*, the treatment to be employed on every occasion. There is no other drug known which can be relied upon so absolutely, or which is so powerful in its action and devoid of marked toxic effect, unless given in enormous doses. Indeed, no fatal case of poisoning has ever occurred from it alone, so far as the author is aware, and he has searched the matter thoroughly

The doses to be used vary with the salt employed to a considerable extent, and depend upon the character of the disease and temperament and physique of the patient. We have already pointed out that the greater the duration of epilepsy is, the greater the difficulty is in effecting a cure; and the length of time which the man has been epileptic should therefore be most carefully reckoned before the treatment begins. Further than this, the frequency and severity of the attacks are to be looked into, and these points are really more important than the actual duration of the ailment; since, if a man has only one fit every six months for twenty years, his condition is far less serious than if he has a history of three or four fits a day for one year. Again, the char-

acter of the attack, as to its violence, may be the most important fact to be regarded; for, if they are violent enough when they come on to endanger life, remedies must be pushed even beyond the point of toleranee. The author has heard a very celebrated physician cause much amusement among his auditors by detailing an instanee of an epileptie who was getting well, and would have recovered *if he had not died.* His explanation was that the man was reeeiving moderate doses of potassium, which were slowly benefiting him, and would have eured him had not a single scvere fit produeed death in the meanwhile.

Another point to be caleulated upon is the condition of the digestion, which the bromide of potassium is peculiarly liable to disorder, and which is sometimes so troublesome as to necessitate the administration of the drug by the reetum in serious cases. Females generally require smaller doses than males, and children of both sexes do not require as large quantities as adults. The dose to be used in the beginning of the treatment, in moderate cases, is about 10 grains thrice a day; and, while this may seem a very small quantity, the writer has found that it ean be rapidly increased in amount without causing the gastrie distress produced by the sudden use of larger doses. Every day may have an additional 10 grains added, until at the end of a week the patient is taking 80 grains each day. There are very few cases which will not beeome eompletely saturated by the drug if this is done; and there are very few in whieh a more rapid eondition of bromism is needed. If, however, the patient has become able to stand large amounts by the prolonged nse of the drug, the amount given is not to be governed by grains, but physiological effeets, and it may be pushed almost to any amount which is borne.

It has been my experience, too, that in chronic epilepsy, with regularly recurring fits, the greatest good is obtained by pushing the drug in ascending doses for one week, and then, for the succeeding week, give only enough to preserve the general effects of the medicament. By doing this the stomach gets a rest and the appetite is not so interfered with. Where the attacks occur only every two weeks this is a particularly useful method, for obvious reasons. As regards the time of day when the drug is to be taken, there can be no variance of opinion. Some writers have directed that it shall be taken always before meals; but this is entirely lacking in advantage, and decidedly fruitful of harm. Medicines to be given so as to affect the general system should be taken after meals, not before, and it is only when a local gastric effect is desired that we use them on an empty stomach, particularly when the substance is as irritant and depressing as potassium. If taken after meals, the appetite is not decreased, but there are few who can take a dose of 10 to 20 grains of the bromide of potassium before breakfast without suffering from anorexia.

It has been held by some that the drug should be taken in minute doses, frequently repeated, in order to keep the patient constantly under its influence. This is an example of therapeutic ignorance, which will be explained when the author speaks of the elimination of the bromide, and possesses the disadvantages of being inconvenient, annoying, and apt to disorder the stomach.

If the attacks have a distinct periodicity, or can be foretold for as much as two hours beforehand, the remedy may be taken in a large dose at this time, and only a few grains given in the intervals. If these attacks are severe, no one should hesitate to use large doses by the mouth and by the rectum on the day of the attack

A very important point to be borne in mind is that the drug often seems to have produced a complete cure, and this results in carelessness in the regularity of administration. The patient should be impressed by the fact that every day passed without a fit is a step forward, and that every fit carries him many steps backward. He should also be made to use the drug in moderation for at least three years after all fits have ceased, and to watch, after that time, for the slightest sign of their return. The quantity taken each day should be gradually decreased, not suddenly stopped short.

It is true, also, that if a recurrence of the fits take place they yield to treatment very much more slowly than before.

Before passing on to the discussion of the other bromides, and the conditions produced by the excessive use of all of them, let us first attempt to place our use of these compounds in epilepsy on the scientific footing already spoken of. In the section on Pathology it was pointed out sufficiently clearly that the seizures known as epilepsy were probably cortical in origin, and the author will go upon this basis here.

There can be no doubt that the bromides act very powerfully upon the cerebrum in the higher animals, decreasing the irritability of the motor centres in these regions to a very great extent. Not only is this pointed to by clinical fact, but the well-known researches of Albertoni [1] seem to prove that such is their action beyond all cavil. This investigator found that the administration of a single dose of the bromide of potassium so lessened the excitability of the motor cells in the cortex cerebri that much stronger stimulation was necessary in order to cause response in the limbs than was normal,

[1] Arch. f. Experimental Path. und Therapie, xv, 256.

and that it was difficult to produce epileptic attacks by means of electrical stimulation of these areas, even when currents were used very much stronger than those which commonly so result. He also found that this lessened irritability was increased still further if the drug was given for several days beforehand in such doses as to thoroughly impress the organism. It is therefore evident that the bromides act directly on the cortical areas, calming the tendency to explosions of nerve-force.

The results of Seppilli [1] have also confirmed those of Albertoni in every way.

An enormous amount of research has proved also that the drug may be doubly useful in reflex epilepsies, not only by its action on the motor cortex, but by its influence on the afferent portion of the nervous system.

The experiments of Eulenberg and Guttmann [2] prove that the sensory paths in the spinal cord feel more powerfully than any other portion of the body the effects of the drug, for they found that if they tied the blood-vessels supplying one limb of an animal, and then injected the bromide into the body, reflex action was abolished equally on both sides, proving that the loss of reflex action does not depend upon the action of the drug on the sensory nerve-trunks. That the loss of reflex action is not due to an action on the motor portion of the cord is proved by the fact that voluntary motion is completely preserved. As these experiments have been confirmed by Lewisky,[3] Bartholow,[4] Purser,[5] and Laborde,[6] there can be no doubt of their truth, and we can

[1] Rivista Sperimentale di Frenatria, fasc. i and ii, 1884.

[2] Virchow's Archiv, xli, 1867.

[3] *Ibid.*, xlv, p. 191.

[4] Bromides: their Physiological Effects. Providence, 1871.

[5] Dublin Journ. Med. Sci., xlvii, 324, 1869.

[6] Archivs de Physiol. Norm. et Pathol., t. i, p. 423, 1868, and Comptes Rendus, t. lxv, 1867.

rest assured that not only does the drug prevent nervous disturbance in the cerebrum, but that it also prevents the peripheral irritation from traveling up to the brain, there to produce morbid excitement.

Apropos of the theory that epilepsy is due to vasomotor disturbance, which has been shown to be unfounded, it may also be added that the bromide of potassium was, and is, believed by some to effect a cure by producing a vasomotor spasm at the base of the brain. There is not one atom of reason in this idea, even if the disease were due to vasomotor changes.

Hammond and Amory[1] have seen the circulation in the brain slowed by the drug, and it has been claimed by Lewisky that if the toes be cut off the blood flows from them more slowly in the poisoned animal than in the normal frog. None of these facts prove vasomotor action, but rather that there is a lessened circulation by reason of the cardiac depression produced by the potassium, which is well known to occur.

An important therapeutic point is to know how rapidly bromide of potassium is eliminated, so that we may know how frequently to give the drug. That it is passed out with only moderate speed is certain, for Rabuteau[2] has seen its presence in the urine one month after the last dose, and Bill [3] has found it two weeks after the use of the drug had ceased. Amory[4] recovered, on the other hand, one-half the amount ingested in the first succeeding twenty-four hours and one-third in the second twenty-four hours. It is evident, however, that it is

[1] The Physiological Effects of Bromide of Potassium, part ii, p. 147. Boston, 1872.

[2] I only know this paper by reputation, and have been unable to find the reference to it. There has been very little work done on this subject on the other side of the Atlantic.

[3] American Journ. Med. Sci., July, 1868.

[4] *Loc. cit.*

eliminated so slowly that doses given three times a day make the patient ingest more than he passes out. That it remains long in the system is proved by the fact that, after repeated doses given to a healthy man, marked somnolence persists for days.

There is one more point to which attention must be called, and that is the fact that when the bromides are taken for any length of time they produce bromism, which, in its moderate or severe forms, produces a mental condition very closely allied to that seen in old chronic epileptics. This condition of the mind should never be overlooked, and the writer believes that the mental changes of epilepsy are greatly increased by its constant and careless administration.

Bromism, or chronic poisoning by any one of the bromides, is often a very troublesome symptom, which has to be dealt with carefully, for if the drug is withdrawn the attacks return. The first signs of this generally are shown by an acne of the face, which may soon involve the whole surface of the body, and, if not relieved, give rise to a condition in which the face and neck become a mass of sores covered with pus. Even when the drug has only been used thus for two or three days, this milder form may occur in those who have an idiosyncrasy to the bromides; and, if the patient be in the higher walks of life, or a woman, it may be impossible to overcome his or her dislike of the drug on this account. It is my custom to give a small quantity of arsenic along with each dose in such patients, or, indeed, in all cases where the drug must be pushed to extremes. The influence which the arsenic exercises elsewhere than on the skin is unknown, but the author is confident that it very strongly acts in protecting the sexual apparatus, and that it also aids the digestion and appetite for food.

It has already been said that the bromide probably aids the disease in producing mental hebetude in some cases, and this symptom very early comes on in bromism. There is often failure of memory, somnolence, loss of spirits, and loss of sexual desire and power. If the drug is used after this, all the powers fail, and the man dies from total extinguishment of all vital action.

The salts of iron, sodium, lithium, nickel, and ammonium have all been used in epilepsy with good results, but, except in certain instances, they fail to act as well as that of potassium, unless given in larger doses. There are several occasions in which, however, each one possesses marked advantages, and may succeed where potassium has failed. In all cases of epilepsy complicated with anæmia the bromide of iron should be employed, but where there is plethora it will generally increase the disease or do no good. Where it acts after potassium fails, the iron is necessary because of its tonic and food effect.

Bromide of sodium, while somewhat less powerful than potassium, is not by any means so apt to disorder the stomach, and is preferable in some cases on this account. It possesses no other advantages.[1]

The bromide of lithium has been highly recommended in intractable cases by Weir Mitchell,[2] who even states that it may be given in one-half the dose of the potassium salt with equally good effects.

The bromide of nickel cures some cases where all other remedies fail, but this is rare. In a series of physiological experiments made by the author some years since, he found it virtually identical with the potassium

[1] Decaisne, as a result of a number of trials, thinks it identical with the potassium salt, save that in large doses it produces constipation, not diarrhœa.

[2] Am. Journal Med. Sciences, October, 1870.

salt in its action, and he has found it useful in about the same doses as bromide of potassium.

The bromide of ammonium is very irritant, and disorders the stomach quite readily. It ought always to be used, when used at all, with some other drug, the ammonium only acting as an adjuvant.

Several authors have tried hydrobromic acid, but it is very much more apt to derange digestion and to produce vomiting than any of the salts. The dose of the dilute acid is $\frac{1}{2}$ to 1 ounce in a tumblerful of sweetened water.

The bromate of potash has been used by Mitchell in not more than 5- to 10- grain doses with good results, but is more dangerous, and scarcely of greater value.

There can be no doubt that in some instances what is known as the mixed treatment is successful where all else fails. This consists most commonly of a prescription in which the bromides of potassium, sodium, and ammonium take part. Why this combination acts better than any one of the salts alone no one knows, but it is certainly a clinical fact.[1]

In other cases still, digitalis, when used along with one of the bromides, seems to carry out favorable results. Indeed, digitalis has for years been used alone in epilepsy with fairly good results, and should always be used in obstinate cases. In petit mal, where bromide of potassium alone so often fails, it is useful, and several English writers, notably Gowers,[2] assert that its best effects are in cases of nocturnal epilepsy. Why this should be the case no one is able to decide, and it would seem doubtful whether it does any more good in nocturnal

[1] As one example of such experience, see article by Erlenmeyer in Centralblatt f. Nervenheilkunde und Psychiatrie, etc., No. 18, 1884.

[2] Nervous Diseases. London.

attacks than in the others. The writer is also unable to explain why it should influence epilepsy at all, for its action on the nervous system is slight, save in toxic amounts, when it lessens reflex action very markedly, first, by stimulation of Setschenow's reflex inhibitory centre, and later by paralysis of the spinal cord. This latter action never occurs, of course, in its medicinal use; but in medicinal doses it may, by acting on the inhibitory centre, allay convulsive tendencies. Probably its chief action is through its circulatory effects, and further study may show it to be efficacious only in those cases where a heart tonic is required.

Another combination very much employed and lauded is the bromide with belladonna, the mydriatic being alone almost useless, but of great antiquity in its use in epilepsy.

Like digitalis and bromide, it succeeds very frequently in petit mal, and indeed seems to be much more successful than the digitalis, but its mode of action is exceedingly doubtful. As the drug acts even more powerfully upon the nervous system than upon the circulatory apparatus, it has been thought that its influences for good depended upon this effect, but the experiments of Seppilli[1] contradict this belief; for he found that, if atropine was given to an animal, the surface of the cortex cerebri responded more readily than is normal to stimulation. Professor Albertoni[2] has also made a series of experiments to determine whether it inhibits the motor powers of the cortex. In his hands, repeated small doses, or one large dose, in no way retard the convulsions produced by stimulation of the brain. Both these investigators are therefore in accord.

[1] Rivista Sperimentale di Frenatria, fasc. i and ii, 1884.

[2] Arch. f. Exp. Path. und Pharm., xv, p. 265.

At one time it was held that belladonna acted on the spinal cord and peripheral nerves under such circumstances, but it should be remembered that we know now that atropine is only of value in relaxing spasm when given in full dose, and oftentimes hypodermically, and that under these circumstances it affects rather the motor-nerve endings than the central nervous apparatus. At the present time those who believe the origin of epilepsy to be dependent on cerebral vasomotor spasm rest the occasional good results from the use of this drug on its vasomotor influence; but there is a good reason for throwing this idea aside, even if the morbid process was really present, namely, that the drug in ordinary medicinal doses raises arterial tension by stimulation of the vasomotor centre, while it only lowers blood-pressure when given in toxic amounts, and then by an action on the blood-vessel walls.

As long ago as the early part of this century, cannabis indica came into notice in the treatment of epilepsy, and is probably of much more value alone than with any other drug. Although it is at present rarely so used, from the writer's own studies he thinks it of value, for he finds that it distinctly lessens reflex action and acts powerfully upon the higher nervous centres in the brain. Its use and value in migraine is undeniably of the greatest importance, and attention has already been called to the fact that several eminent neurologists believe epilepsy and migraine to be very closely allied. The effect on the circulation is almost *nil*, and its influence is solely expended on the nervous system. The deep sleep produced by it, even in moderate medicinal doses, is not only deep but prolonged, and it undoubtedly quiets the sensory nerve-trunks all over the body, as well as the sensory side of the cord. Indeed, it seems

to resemble the bromides in its action quite closely. It should be given in doses of $\frac{1}{4}$ to $\frac{1}{2}$ grain of the solid extract or 20 minims of the fluid extract. Like the bromides, too, it is very rarely capable of producing serious results, and there are no cases of a fatal character reported from its overuse. To illustrate its slight lethal power, it may be stated that the author has injected into the jugular vein of a dog not less than 35 cubic centimetres of a fluid extract, the dose of which was physiologically active at 8 minims in man before producing death.

Gelsemium sempervirens is an American plant whose praises, in almost every disease, have been widely heard. Its influence alone is almost worthless, for it possesses no power over the cerebral centres whatever; but in combination with cannabis indica it makes a very useful agent, as it quiets any excitement in the spinal cord and depresses its conducting power, while the cannabis indica, in its turn, quiets the cerebrum. The dose of the tincture is 20 drops, but it should be remembered that it is as poisonous as the other is innocuous.

Owing to the soporific influences exercised by opium it has been very frequently tried, with success and failure as a result. It certainly has not taken any rank in the list of remedies, and this is a deserved withdrawal of professional favor. It increases reflex activity very commonly, and seems to affect the intellectual areas of the cerebrum rather than the motor portions, although Seppilli's experiments show it to exercise a decided depressant influence over these areas. Combined with gelsemium it may, perhaps, be employed, but only when nothing else is at hand or all other remedies have failed. If it is so employed great care is to be used, and it

should not be forgotten that both drugs kill by respiratory failure. When used in "status epilepticus" it often does the most good in relieving the spasm, but it must be employed in large doses, and if the succeeding coma of epilepsy has added to it that of large doses of opium death may ensue.

The employment of zinc has been very greatly recommended for many years, but has found little favor of late among the profession generally. It has been stated that it quiets the cerebral cortex, the medulla oblongata, and spinal cord, and in this way cures the attacks. This is, however, merely clinical evidence, and has no experimental proof to support it.

Even its most sanguine supporters confess that its range of usefulness is generally in those cases where the bromides succeed, and agree that its powers are much inferior to these compounds. The dose of the oxide is 3 to 7 grains twice or thrice a day, and even in this amount may cause nausea and vomiting. The citrate is more soluble, and is better borne by the digestive apparatus. Its influence over the disease is probably the same, as is also true of the lactate, which was so largely used by Herpin (*loc. cit.*), and which is, so far as my reading goes, the best salt of zinc to use.

Nitrate of silver was brought into use long before the value of more recent drugs was known. Every one is agreed as to its lack of curative power, and no one has ever claimed good results from it save when it was used constantly for a long time. As the drug is eliminated very slowly, it rapidly accumulates, and argyria soon comes on. It may be used, after all else fails, in doses of $\frac{1}{6}$ to $\frac{1}{4}$ grain, thrice a day, after meals, and the mucous membrane of the inside of the lips and the conjunctiva should be carefully watched for

the early signs of chronic silver poisoning. We certainly have no knowledge as to its influence on the nervous system, and, if it acts at all, it must be by some alterative influences rather than by any other means.

Nitro-glycerin is to be employed rather in petit mal than in haut mal, in the dose of 1 drop of a 1-per-cent. solution once, twice, or thrice a day. Our knowledge of its effects, so far as its curative influences are concerned, is very slight, but it really seems to benefit some cases. Its action is very fleeting, and one is inclined to believe that it influences the brain very little except it be taken just before an attack is expected, or where the cardiac action is defective. Its great lethal power should never be forgotten.

The use of the nitrite of amyl is not for the purpose of directly curing the disease, but of warding off impending attacks, the warning of which is given by an aura of slow progression. The author has pointed out already that it increases the severity of petit mal. In epileptics who have a prolonged aura we may use nitrite-of-amyl pearls, which consist in small glass bulbs containing a few drops of the drug. As the aura comes on, the patient should break one of these in his handkerchief and inhale the drug, thereby putting aside the attack.

The influence which the drug exerts upon the brain is secondary rather than primary, and is probably dependent on its action on the blood or circulation. Its influence on the spinal cord and nerves is much more marked and direct, and it is most certainly a very powerful spinal depressant. As its influence over unstriated muscular fibre is very great, it affects the vasomotor system very powerfully, and those who think that epilepsy is due to vasomotor spasm at the base of the brain point to the effects of this drug as a proof of their

hypothesis. Such reasoning is not, however, neeessarily correet. The writer is inelined to believe that the nitrite of amyl puts aside an attack by a sudden shoek to the nerve-eentres, whieh diverts them, so to speak, from their intended diseharge, very mueh as a ligature stops an aura. When we remember that the drug acts instantly, and converts nearly all the oxygenating blood of the body into a non-oxygen-earrying fluid by reason of the nitrite-oxyhæmoglobin produeed, the sudden change in the eerebral nutrition and life is most marked. In the "status epileptieus" it is of great value in stopping the seizures, and may be used under these eireumstanees in heroie amounts applied at intervals to the nostrils. In the tonie spasm, if it be severe enough to stop respiration, it should be remembered that, as the drug is not inhaled, it is absolutely worthless. It is only when a moment of relaxation oeeurs that it does its work.

As a general rule, the nitrite of ammonium or sodium, whieh are more prolonged in their effeets, should be used to supplement the amyl salt.

Gowers states that nitrite of amyl does good by flooding the brain with arterial blood. How sueh a statement ean be made by any one is amazing. Of all the drugs in the world nitrite of amyl produces exaetly the opposite ehange, as Gowers should be aware from his own use of the substanee.

The use of anæsthetics in epilepsy is virtually useless, and, in some eases, dangerous, for ether is too slow in its effeets, and may, by its irritant vapors, inerease the tendeney to laryngeal spasm or eause lung eomplieations. Further than this, if uræmia is the eause of the fit, and this faet is unknown in every ease until it is examined, the ether may increase the inflammation of the kidney very seriously.

Chloroform, though it acts much more rapidly, may cause sudden cardiac failure, and both drugs may increase the post-convulsive coma very greatly.

In "status epilepticus" they may be used, as, in such cases, the convulsion must be stopped at all hazards, and the preference should always be for amyl nitrite.

The iodide of potassium is entirely useless in epilepsy, unless it is due to syphilis, when it is of the greatest service. Indeed, the bromide and all other drugs should be set aside, while it is pushed to the utmost. As is well known, syphilitics usually bear the drug extremely well, and the writer knows of one instance where no less than 800 grains were taken every twenty-four hours, with rapid improvement as a result.

This point is strongly insisted upon by all therapeutists and syphilographers, notably among whom stands Fournier.[1]

Where the convulsions are due to a gumma the iodide of potassium is, however, too slow in its action, and should be replaced by mercury in order to break down the growth without delay, lest a seizure end the scene by asphyxia or some similar accident.

Some difference of opinion exists as to the usefulness of iron in epilepsy. Several very eminent clinicians have asserted that it always makes the attacks worse and, therefore, does more harm than good.

The writer thinks that, like everything else, iron is no more to be given in every case than is a dose of oil, but that where there is plethora it is harmful and where there is malnutrition and anæmia it does good. Over the disease itself it really has no effect at all, except through its action on the general system.

Chloral hydrate is a remedy which has been only

[1] L'Union Médicale, 1875, et Annales de Dermatol. et Syphilog., 1880.

partly tried in epilepsy, and its usefulness is not as yet determined. It possesses the marked disadvantage, as compared to the bromides, of being a very fatal poison, which is an important fact to be borne in mind by the physician when giving it to a patient, whose mind, already weakened by the disease or naturally stupid, may forget and take too much. Its physiological action indicates, much more fully than many other much more lauded remedies, that it may be of value, since it exerts its chief influence on the motor pathways of the spinal cord and quiets the motor portion of the cerebral cortex, and also produces sleep. Seppilli [1] has proved this, too, by direct experimentation after the method employed by Albertoni. Its use, combined with one of the bromides, is often accompanied by the most desirable results, and should be tried at all times unless some cardiac complication forbids it. It may disorder the stomach, and should, like the bromides, always be given well diluted and after meals.

Of the more recent remedies, *antifebrin* certainly stands in the foremost rank, and bids fair, in some instances, to rival the bromides. Prof. Germain Sée and the writer have reported cases which obtained very marked relief from it, and more recent investigators have done likewise. The author's experience with the drug has been that it exerts its chief benefits in chronic epilepsy. At least, if a child were brought to the writer with a beginning epilepsy, he would use the bromides, but, if the disease was chronic, the antifebrin. In the cases seen by the writer the patients were adults, and had very marked mental failure, one of them being virtually idiotic and a sufferer from two to six attacks every day Bromides had lost power over them, and

[1] Rivista Sperimentale di Frenatria, fasc. i and ii, 1881.

antifebrin certainly acted most marvelously. Thus, in one case the fits fell from twenty-one to four a week, when the patient was lost sight of. It should be used in the form of powder, on the tongue, in the dose of 8 grains thrice a day, with a little sugar.

Mabille and Ramadier [1] have found acetanilide very useful, also, as has Leidy,[2] who found benefit produced by the drug in 14 cases out of 26, and also that the drug influenced more favorably still petit mal.

Bowsnyoi [3] treated 9 epilepties in this manner, using doses ranging from 3 to 30 grains. He believes the drug to be inferior to the bromides, both in its control of the disease and in the depressant effects which it sometimes produces. He found, however, that large doses generally controlled the attacks.

On the other hand, Salm,[4] in Jolly's clinic in Strasburg, gave the drug in doses of from 1 to 3 grammes a day, and not only saw no benefit, but some of the cases became worse.

Antipyrin has also been pushed forward as a remedy, and, as the physiological action on the nervous system is virtually identical with antifebrin. They will be spoken of together.

Antipyrin was recommended by Lemoine,[5] in 1887, in certain forms of epilepsy, but condemned in most cases.

In those who suffer from menstrual epilepsy, so called, or in those in whom the attack is produced reflexly by the presence of intestinal parasites, the drug does good.

Lemoine also found it very useful in those cases

[1] Soc. Méd. Psycholog. Séance, Juin 27, 1887.
[2] N. Y. Med. Journal, vol. i, 1888.
[3] Centralbl. f. d. Gesammte Therapie, March, 1888.
[4] Neurol. Centralblatt, 1887.
[5] Gazette Méd. de Paris, December 24, 1887.

associated with migraine. In these cases the results were better than with the bromides, but in the idiopathic, simple varieties it was useless. Mairet and Combemale [1] have used the drug in epileptiform mania with very satisfactory results.

In children suffering from frequently recurring epileptic attacks the presence of worms should always be looked for, and, when found, they should be expelled as rapidly as possible. If they are the *Oxyuris vermicularis*, the best remedy, by far, is the injection of a strong infusion of quassia of such a strength that there is 1 to 2 ounces of quassia to each pint of water.

In girls, where the removal of the worms from the rectum is not followed by relief, a careful examination of the vagina should be made and quassia employed in somewhat weaker solution as a vaginal wash, as, very commonly, intense inflammation is here present, produced by migratory movements of rectal parasites. If the quassia is unobtainable in any case, a saturated solution of chloride of sodium may be employed.

To complete the consideration of the treatment of epilepsy, the writer must add the information which we possess in regard to remedies which, while highly recommended by one person, have been wholly or partly found useless by the majority of the profession.

Foremost among such agents stands curare, a substance whose physiological action makes it about as fit for the treatment of this disease as so much sawdust or equally valuable matter. Every one knows that this drug paralyzes the peripheral motor nerves in the muscles long before it affects, to any extent, the rest of the organism, and it is evident that its anticonvulsive action can only rest on the theory that it blocks the pathway

[1] Gazette Hebdomadaire, December 23, 1887.

to the muscles in such a way that the impulses cause no contractions in them. Even supposing that curare could be given in large enough dose to obtain a full physiological effect, it could in no way prevent anything more than the outward evidence of an attack; and, as the injury lies not in the muscular contractions, but in the central nervous storm, its uselessness is apparent. Even its most vehement supporters are unable to adduce any remarkable results from its use.

Kunz[1] has used it in 80 cases, and seen, so he says, 6 radical cures and several cases of partial relief. Thiercelin, in 1861, found it of value when combined with other drugs, or, in other words, it was useless in itself; and Benedikt, in 1866, reported that it lessened the frequency of attacks, but did not cure. In the same year Mundt declared it useless, and the year before (1865) Voisin made a like assertion. Still more recently, Bourneville and Bricon[2] have tried its anti-epileptic virtues. They administered curare hypodermically to 33 epileptics, but obtained only one cure (?), although in some instances it was given for as long as six months at a time, and they therefore believe curare virtually useless for such purposes. Further, it is hard to imagine a more lethal drug with less medicinal power for good.

Apomorphia has been used by Vallender[3] hypodermically with some good results, but it has certainly not obtained general favor in the profession.

Cocculus indicus, in the form of a tincture, has been employed by Hamhusin[4] in the dose of 10 drops with asserted good results, but its value certainly needs much more thorough tests than any one observer can employ,

[1] Ber. d. 51 Verhandlung deutsch. Naturf. und Aerzte in Cassel.
[2] Arch. de Neurologie, March, April, and May, 1886.
[3] Berliner klin. Wochenschrift, 1877, xiv, 185.
[4] Bull. de l'Acad. de Méd. de Belgique, 1880.

and its use is still *sub judice* in consequence. He found, however, that the system became rapidly accustomed to the drug, and that it could be increased day by day until 150 drops at a dose could be borne.

Picrotoxine is also, in all probability, a useless remedy, but has been recommended by Planat, Conyba,[1] and Hamburgin.

Osmic acid has been quite thoroughly tried by Wildermuth,[2] either in the form of the acid in water or, more latterly, as the osmate of potassium, the dose being in twenty-four hours about $\frac{1}{4}$ grain. In 10 old and chronic cases he reached no result in 7 of them; in 2 the attacks became less frequent, and one previously desperate case recovered. Three later cases gave in two instances quite marked amelioration and in one case no benefit.

The treatment of epilepsy by borax has not received very wide recognition. Perhaps the most thorough studies of its effects have been those of Gowers,[3] in England, and Folsom,[4] in America. It would seem that some cases which are obstinate under ordinary treatment are benefited by it, but it certainly is not to be commonly employed. The doses are generally about 15 grains *ter die*.

Several clinicians have proposed the use of electricity in the treatment of epilepsy, and have attempted to galvanize the brain by placing a pole on each side of the temples or on the forehead and occiput. Still others have tried it by placing a pole on the spine and one on the vertex. Rockwell professes to have benefited cases by this means, but it is extremely doubtful if his results

[1] Jour. de Méd. et de Chirurgie prat., 1880, 214.
[2] Bulletin Gén. de Thérapeutique, October 25, 1884.
[3] Epilepsy. London.
[4] Boston Med. and Surg. Journ., February 18, 1886.

were not due to coincidence or imagination. Electricity resembles water in the law that it always travels in the direction of least resistance, and, this being the case, it is evident that the current passes through the integument over the skull, and not through the bone and cerebrum inside of the cranium.

A practical proof of the failure of the attempt is that the slightest current applied, in reality, to the brain elicits a response, while in the method just mentioned this never occurs.

Having spoken of the drugs which may be given to epileptics, let the writer draw the attention of the reader to those which may not be used. There is a very large amount of reliable evidence, both experimental and clinical, that *quinine* should never be employed where it can be avoided. Thus, Seppilli,[1] in his researches, found that it increased the irritability of the cerebral cortex, and Briquet [2] has asserted that it is a direct cerebral stimulant. That toxic doses of quinine may provoke epileptiform convulsions has been proved by Jakowbowich,[3] who has seen them in dogs and in other animals, and Brown-Séquard and Albertoni [4] have noted that cinchonidine and quinine always increase the number of attacks in epileptics.

Salicylic acid, too, has an effect upon the brain very closely allied to that of quinine, and should always be used with care in epilepsy. Prof. Germain Sée [5] has pointed out also that large doses produce violent epileptiform convulsions in the lower animals.

Strychnia, while its chief effect is to heighten the

[1] Rivista Sperimentale de Frenatria, fasc. i and ii, 1884.
[2] Traité Thérapeutique de Quinquina. Paris, 1855.
[3] Rev. des Sciences Méd., 1873.
[4] Archiv f. Experimental Path. und Pharm., xv, 278.
[5] Bulletin de l'Acad. de Méd., 1877.

activity of the spinal cord, also, according to Seppilli, increases the irritability of the cortex, and should be employed only in particular cases. The same writer also found that absinthe and picrotoxine acted detrimentally in increasing the excitability of the motor zone.

Bleeding the patient in epilepsy, unless there is particular evidence of cerebral congestion, which is exceedingly rare, is harmful rather than of value. Orschansky[1] found that removal of one-seventh of all the blood in the body by the femoral vein did not lessen the irritability of the cortex, and Minksowsky [2] ligatured all the blood-vessels going to the brain without decreasing its excitability. We know also that cerebral anæmia produces epileptic attacks.

A very important point which is constantly brought before the physician who is treating epilepsy is that of diet. Nearly every patient inquires what he shall eat, when he suffers from this disease. So far as the writer is aware, very few researches of a thorough character have ever been carried out on a large scale to determine the foods which may or may not be ingested. Of course, nearly every one of us know from our personal experience that red meats are hurtful, particularly in children. Curiously enough, the influence of diet, in one research covering a number of cases of chronic epilepsy, seemed to be of little moment. Thus, Merson[3] examined 24 such cases, putting 12 of them on a purely vegetable and 12 on a purely nitrogenous diet. The result, after this had been continued for two months, was that the vegetarians had had a few less fits than the others, but the difference was so slight as to be almost of no weight in determining the question. Some authors at the present

[1] Quoted by Seppilli. [2] *Ibid.*
[3] West Riding Lunatic Asylum Reports, 1875.

day believe this opinion as to the harmfulness of meats to be erroneous.[1]

The operative treatment of epilepsy divides itself into two classes,—that concerned with the removal of lesions in the brain, and that which deals with the removal of peripheral irritations, such as adherent prepuce or growths and nerves. The writer will first speak of those forms of epilepsy due to centric causes among the cells themselves, such as abscess, tumor, pressure, embolism, and thrombus. He has elsewhere pointed out that these changes are productive of epilepsy, and it is unnecessary for him to go over them again in this respect.

Notwithstanding the antivivisection laws of England, curiously enough she stands pre-eminent in cerebral surgery; and, so far as is known, the first successful operations for abscess and tumor of the brain were there performed in 1884, by Mr. Godlee. In much the same manner as abdominal surgery suddenly stepped to the front and became a very popular means of relief, so this new field has been largely gone over by surgeons in all parts of the world, and, it may be said, has been remarkably successful, considering the limited diffusion of the knowledge of cerebral localization which has heretofore existed. Indeed, the failures, in the majority of cases which have failed, have depended on the lack of experience in the operator rather than on the gravity of the operation itself. Probably no one in the world is at present so widely known in this branch of surgery as Victor Horsley, of London, both because of his skill and his researches. On a recent visit to England, where he was good enough to let the author see something of his work, the writer was greatly impressed by the fact that his experimental investigations were always used

[1] Gowers, in his book on Epilepsy, is one of these.

for the furtheranee of his professional work, and that he represented, *par excellence*, the logieal physician and physiologist.

None who have seen men familiar with brain localization operate ean do so without being impressed with the faet that the present century has given birth to still another medieal triumph. Aeeording to the most sueeessful operators of the day, the most striet and careful antisepsis is preserved during the operation, which eonsists in first shaving the entire head, and then bathing it with a solution of the bichloride of mereury or soap. Horsley uses earbolic-aeid solution. The area of the eortex involved is now traeed out on the skin, and the operation then eonsists in forming the flap and trephining as usual. The majority of surgeons do not use the earbolic-acid spray, but Horsley insists that it is absolutely neeessary, informing me that in every ease where he has operated on monkeys without the spray death has oecurred, whereas where it has been used they invariably reeover. The dura is next laid aside in the form of a flap in mueh the same way as the sealp, but the greatest possible eare is exereised, lest, in the movements of the operation, any pressure be made on the cortex, as this is very often followed by paralysis of the limb supplied by that area, at least for a time. Care is also to be shown that no part of the eortex at any time beeomes dry, and if the operation is prolonged the flaps should be now and again laid over the part to moisten it. In some instances the tumor or abseess is subeortieal, rather than eortieal, and it becomes neeessary for the operator to make either exploratory incisions or digital examinations for it. The hæmorrhage from the brain is not so violent as might be supposed, and ean be eontrolled by eompresses, or, where large surfaee-vessels appear, by torsion or ligature.

The most troublesome oozing is from the diploœa, and this may be stopped by a mixture of bees-wax and gum-benzoin, which is melted in a test-tube and boiled, in order to sterilize it before it is used. When it hardens again, it is rubbed over the bleeding spot, and checks all hæmorrhage; nor does it influence the recovery of the patient in any way whatever. The following successful case, operated on by Dr. W. W. Keen, is of interest, as showing the value of cerebral localization as a scientific fact, and its use in relieving humanity, as well as the enormous size of the growth:—

A man, aged 26 years, was injured by a fall from a window at the age of 3, his head striking against a brick. A superficial wound was made in the scalp, but no trouble was experienced from the injury until twenty years had elapsed, when epilepsy developed itself. At the same time there was aphasia and paralysis of the right arm and leg; these last symptoms, however, soon passed away.

The operation was performed December 15, 1887, and the tumor removed measured $2\frac{7}{8}$ inches in its long axis and $2\frac{1}{8}$ inches in its short axis. It was $1\frac{3}{4}$ inches thick. It extended from the fissure of Sylvius into the first frontal convolution, and from near the fissure of Rolando into the bases of the three frontal convolutions. The weight of the tumor was 3 ounces and 49 grains.

The patient did remarkably well for two days after the operation. On the third day marked symptoms of pressure showed themselves, and this was ascertained to be due to the presence of a large clot of greater size than the tumor itself. This was carefully removed, and all went well for ten days, when pressure symptoms were again observed. There was also severe diarrhœa and a temperature of $104\frac{1}{2}^{\circ}$ F. The presence of pus

was suspected, and the wound was accordingly re-opened. This resulted in a hernia cerebri. Later there were several less severe attacks of diarrhœa, accompanied by more or less rise of temperature. The hernia was somewhat persistent, and, in order to facilitate the process of healing, skin-grafts were made from the arm. The wound was dressed with biehloride gauze for eight weeks, but there was no evidence of absorption, nor was the diarrhœa attributable to it. The spray was not used, but all other antiseptic measures were employed. The wound healed completely, and the man recovered not only from the operation, but from the epilepsy.

The operation of trephining for any traumatic condition of the skull in epilepsy has now, as has already been said, reached a point of acknowledged value.

Briggs [1] has recorded 30 cases of this character, of whom 25 were cured, 3 were relieved, 1 was not benefited, and 1 died. Again, the statistics of Walsham,[2] giving 130 such cases, though they are not quite so favorable as those of Briggs, are of great value, for 75 of them were completely cured, 18 were improved, and 30 died. Seven were not benefited. It will be seen that out of 160 cases there were 100 cures, 21 improvements, 7 failures, and 31 deaths. It is evident, therefore, that operative proceedures in such cases are more fruitful (62.5 per cent.) than any form of medication in any form of the disease. The necessity for operating becomes very evident if we can rely on the assertion of Garmany[3] that 50 per cent. of all cases of frontal traumatism become epileptic.

A method of treatment which is quite heroic is that

[1] American Practitioner, July, 1884.
[2] St. Bartholomew's Hosp. Rep., vol. xix.
[3] Trans. Ninth. Internat. Med. Congress, 1887.

adopted by Alexander,[1] an Englishman, namely, ligation of the vertebral arteries. The operation consists in making a linear incision opposite the lower end and outer side of the external jugular vein "for 3 inches," going down through the fascia between the anterior scalenus and longus colli muscles, and in tying one or both vertebral arteries at the sixth cervical vertebræ. His results in 21 cases are, in his opinion, sufficiently good to decide the value of this line of treatment, but we cannot agree with him. In 9 of the cases, up to the time of his report, no fits have occurred for a very long time and in 8 cases they are becoming less frequent; in the remaining 4 not much change has been noted,—indeed, one died in a paroxysm. He is careful to state that he has little confidence in the operation if the attacks are chronic, but denies any serious after-effects. Practically, however, the surgeon would hardly feel justified in performing such an operation until the disease had been treated by all other means and so had become chronic, and even then might hesitate for a considerable time, simply because the risks to be run seem about equal with the chances of relief. The reasoning by which the procedure is arrived at is that the convulsions depend chiefly upon alteration in the nutrition of the medulla oblongata, and by this operation the circulation is changed sufficiently to produce a cure. It is hard to see why it should not make a case worse rather than better.

There are several cases on record in which removal of the testicles has resulted in the cure of epilepsy. As long ago as 1855, Dr. McKinley, an American, reported several such instances,[2] in 2 of which disease of the

[1] Brain, 1882–83, p. 170.

[2] American Med. Gazette, July, 1855.

testes occurred in epilepties who were cured when the glands were removed. In the other cases the operation was performed with the cure of the epilepsy in view, there being no disease of the testicles. Two of these occurred in the practice of Dr. White, of Tennessee, 2 in the practice of Dr. Talbot, in Missouri, and 1 in the experience of Dr. Hacher, of Louisiana. Another case is that performed by Holz at the request of Frank.[1] All the American cases are recorded in McKinley's paper, and in all of them the cure was accomplished.

Bacon[2] has also very much more recently revived castration as a means of cure, particularly in insane males who were masturbators.

Operative surgery has even interfered with idiopathic epilepsy, for we find the records of several cases where nerve-stretching has been performed with more or less satisfactory results. Thus, Gillette[3] reports an instance of a woman with congenital epilepsy, in whom he stretched the median and ulnar nerves in the upper third of the arm, with the effect of decreasing the attacks from 90 to but 18 per month in the course of less than three weeks.

Much has been written and spoken by able men as to the wonderful relief afforded in some cases of epilepsy by the removal of peripheral irritation, which, in many instances, was very obscure and ill defined. Like every other measure of relief, it is probable that a greater usefulness was claimed for it than was deserved, and probably many persons are lacking foreskins, ovaries, or clitoris by reason of the search for peripheral enemies of health. No one can deny, however, that cases of reflex

[1] Praxes Medical Universal Preceptor, vol. ii, chap. xi.
[2] Journal of Mental Science, Oct., 1880, p. 470.
[3] Le Progrès Médical, February 5, 1881.

eharacter do exist, and that very frequently their removal results in recovery. In the case of adherent or inflamed prepuee, partieularly in children, this should be removed or every care taken that all foreign matter, sueh as urine and smegma, is kept out where the operation is not permitted. Cireumeision is an operation whieh is not only simple in its performanee but laeking in danger, and is always justifiable, if only for the sake of the attempt at cure. Very frcquently, as has already been said elsewhere in this essay, masturbation is thus done away with and an element of nervous relief gained. Where masturbation occurs in epileptie girls, clitoridectomy may be performed with suecess, or even the ovaries extirpated, as has already been detailed in one case.

Closely allied to this is the influence which Charcot has shown to be possessed by pressure on the ovaries in some cases of epilepsy. Certain epilepties immediately bccome convulsed if pressure is made, and in these there is generally very marked ovarian tenderness. If the tenderness does not pass away by ordinary means, the ovaries should be removed by the ordinary operation.

Sometimes, after amputations of the fingers or an extremity, neuromata form on the ends of the severed nerves, and they become entangled in the cieatrix, giving not only much pain, but also producing epilepsy. The surgeon should here remove the growths or free the nerves from the scar. In some manner epileptogenie zones sometimes develop, and should be excised.

For many years the operation of trephining arcas where depression seemed to exist has been eustomary in very obstinate eases of epilepsy, and some surgeons have gone so far as to trephine in any case, hoping in some indireet way to relieve the morbid process.

The writer does not, however, see that this comes within the confines of modern surgery, bold though it be; for, unless some real reason for trephining exists, the operation is too severe to be performed in the dark.

One may also mention here the treatment suggested by Marshall Hall, namely, tracheotomy. Of course the absolute uselessness of this operation as a cure for epilepsy is known to-day, but Hall believed that the convulsion was due to "carbonized blood," produced by the asphyxia brought on by closure of the glottis.

Duration and Number of Epileptic Fits and Mortality.—Epilepsy is absolutely unlimited, except by death, in the length of its existence in a given case. No age which the patient may attain rids him of his Nemesis, which follows him to the grave whether he be in the prime of life or broken down with years. Of course any given attack may cause death, as has been shown under the head of prognosis, but otherwise no change may occur in the vitality of the patient, unless the fits are violent or frequent enough to cause exhaustion.

An exceedingly interesting case of this character has been recorded by Prichard[1] of a man of 71 years, who had been a confirmed epileptic for fifty-seven years. He worked at baskets for a living, and would often have as many as four or five fits a day, interrupting his work. On several occasions he suffered from as many as thirty in one day; indeed, it was estimated that in all he had in his life no less than sixty-five thousand (65,000) fits. He seemed but slightly dulled by them, and would go on working till another came on. His wife and himself became so accustomed to their occurrence that they came to be regarded as part of their daily life. On one occasion, in his seventy-first year, however, the man

[1] British Med. Journ., April 28, 1860, p. 319.

failed to return to consciousness, and upon examining him he was found to have suffered from an apoplexy, from which he died. At the autopsy the dura mater was natural, but the arachnoid was opaque. The ventricles were filled with blood from the rupture of the artery. In the falx major anteriorly was a considerable plate of bone, $1\frac{1}{2}$ inches long, tolerably thick, and flat on the side toward the membrane, while on the other side it was markedly indented by the convolutions. In the upper part of the convexity of the left hemisphere were two round deposits of bone, as large as nuts, under the arachnoid and in the pia mater, pressing down into the substance of the brain, which was much softened about them. There was a single, much larger deposit of the same shape below, and another was attached to the petrous portion of the temporal bone by a pedicle, and occupied a cavity in the substance of the middle lobe.

An extraordinary number of fits may occur in a brief space of time without causing death, or even very great exhaustion; at least, in some cases. A very good example of this fact is that of a case reported by Newington,[1] which is as follows: On the twentieth day of the month, at 9 A.M., the fits began in the woman under his care. By 9 P.M. the same day she had had 274 fits, and by 9 A.M. on the 21st she had 384 more, or 622 fits in twenty-four hours. This makes a rate of 1 nearly every minute. By 9 A.M. on the 22d she had 400 more, by 9 A.M. on the 23d 525, by 9 A.M. on the 24th 395, and from 9 A.M. on this day to 9 A.M. on the 25th she had 214 fits.

Altogether, she had 2156 fits in five days, and yet survived, being fed by the rectum. This seems almost incredible, but the reporter is evidently reliable.

[1] Journal of Mental Science, 1877, p. 89.

Delasiauve[1] has seen a single patient have 2500 fits a month, and Althaus[2] records the case of a boy who had 1350 in the same space of time. Leszynsky[3] also reports the case of a woman, aged 46, who had 688 convulsions in seventy-five hours, ending in death. In a case of *état de mal epileptique* recorded by Bourneville[4] the convulsions occurred as follows:—

1st day, 20 fits; 2d day, 45 fits; 3d day, 22 fits; 4th day, 27 fits; 5th day, 12 fits.

Doxwell records the case of a male, aged 20 years, who frequently had 200 to 300 fits per diem, and in the last year of his life 21,800 fits.

The interval between the fits is very variable, but all statisticians agree that the most common interval is from one day to one week.

A German observer,[5] Snell, has studied the mortality of insane epilepties in order to compare the death-rate in this class with that of pure insanity, and he finds that insane epileptics have a higher death-rate, generally dying at or before the age of 33 years. The causes of these deaths, as enumerated by him, show that the greater mortality depends on the accidents of the fit than the disease itself, for coma due to asphyxia is very commonly assigned as the cause. Out of 100 cases examined Snell found death due to phthisis in 31 cases besides the causes just mentioned.

Chapman[6] has made studies from the records of asylums to determine the difference in mortality between epilepties in whom the disease had been acquired and

[1] Traité de l'Epilepsie, Lausanne.
[2] Epilepsy, Hysteria, and Ataxia. London, 1866.
[3] N. Y. Medical Journal, Mar. 21, 1885, p. 321.
[4] Bourneville, Etat de mal Epileptique, 1873.
[5] Snell, Zeitserift f. Psychiatrie for 1875.
[6] Jour. Mental Sci., Apr. 1880, p. 15.

those in whom it was congenital. He finds that the acquired form is much more fatal than the latter, and much more so in females than in males,—twice as much so in the congenital form, but three or four times as much in the acquired disease. In the researches of Martin,[1] of Boucher and Cazanvielh,[2] the first-named observer working in the Salpêtrière, and whose labors have been quoted when speaking of the influence of heredity in producing epilepsy, it was found that virtually all children born of epileptic parents are epileptic or dead before puberty.

These results in France have been duplicated in England by Althaus,[3] who has collected statistics of six periods of five years, as follows :—

Deaths.	Periods.	Per Cent. of Nerv. Dis.	Per Cent. of All Dis.
1838–42, ...	5,585	2.66	.32
1843–44, ...	.....	....	..
1845–51, ...	8,667	3.62	.43
1852–56, ...	10,339	4.01	.49
1857–61, ...	11,689	4.31	.54
1862–66, ...	12,359	4.21	.51
1867–71, ...	12,290	3.98	.49
30 years.	60,929	3.86	.47

He also found that the mortality of males to females in epilepsy is as 2.13 to 1.84.

The statements of most writers that the mortality of epilepsy is very low, and which have been given in this essay as the writer's own belief in the matter, have recently received strong contradiction at the hands of Worcester,[4] an American investigator. He examined

[1] Annales Méd. Psychologique, Nov., 1878, to Sept., 1879.

[2] De l'Epilepsie considérée dans ses Rapports avec l'Aliénation mentale.

[3] Nervous Diseases, p. 222. London.

[4] Med. Record, 1888, 33, 467; also see Abstract in Amer. Jour. Med. Sciences, July, 1888.

the statistics of the Michigan Insane Asylum for the past twenty-eight years, as well as those of fifty-five other asylums, fifteen of which give statistics for their entire period of operation. The results show that 20 per cent. to 30 per cent. of the epileptic inmates die of epilepsy, the rate being often much nearer the latter figure. This is a much larger number than the total death-rate of the individual asylums, and shows, therefore, that not only is epilepsy a very fatal disease, but that many more epilepties die from it than from all other causes put together. Worcester admits that the inmates of asylums are generally cases of unusual severity, but on the other hand points out that they are better taken care of, and have greater security from accidents, so that he believes these figures really do represent the death-rate. On the other hand, if it were true that epilepsy is so fatal, it is remarkable that so few deaths occur in the ordinary practice of the average physician and neurologist.

The cause most commonly producing death, other than traumatisms, is, according to Leszynsky,[1] in every sixteen deaths of epilepties out of twenty due to status epilepticus.

Proportion of Epileptics to Population and the General Distribution of the Disease.—The number of epilepties to the population of a country forms a very interesting study. Lunier[2] has investigated the subject in France and Tigges[3] in Germany, in one province, that of Mecklenberg-Schwerin. Further studies are those of the census of the Rhine provinces,[4] and those of Meynne[5] in Belgium. Lunier draws his results from the men

[1] N. Y. Med. Journal, 1885, Mar. 21, p.321.
[2] Annales Méd. Psychologique, March to November, 1881.
[3] Zeitschrift f. Psychiatrie, Bd. XI, Heft. 4.
[4] Centralblatt f. Nervenheilkunde, September 5, 1882.
[5] Topographie Méd. de la Belgique, Bruxelle, 1865, p. 101.

examined for the conscription, of whom there were 1,458,740, between 1873 and 1877. In this number there were found 2398 epileptics, which would give the proportion among the inhabitants of France as 16.44 to every 10,000. The objection to these figures is that the cases were all between 20 and 21 years of age; so Lunier has endeavored to eliminate this source of error, and as a result puts the proportion at 16.44 in every 10,000 between the ages of 10 and 40 years.

The census made by Tigges showed that the proportion of epileptics in the town of Schwerin was 1 to every 855 persons, showing the disease to be quite prevalent, comparatively speaking. The studies made in the Rhine provinces were to discover the proportion of mentally unsound epileptics to those who were mentally sound.[1] It was found that those who had mental failure numbered 807, or 23.3 per cent.; while those sound equalled 2653, or 76.7 per cent.

In Mecklenburg-Schwerin the number of epileptics and idiots is virtually the same, for there were found to be 639 epileptics and 658 idiots. Out of 639 epileptics there were 167 idiots, or 25 per cent.

In Wurtemberg the proportion of epileptics to healthy persons is 0.93 per 1000, and in Belgium according to Meynne[2] it is 0.9 per 1000, which is virtually the same.

Hirsch[3] states that the proportion for Southern Europe is 1.5 for 1000, while in France it is 1.6 per 1000. The same authority places the number in Italy at 2.4 per 1000.

Complications.—The various complications which may arise in epilepsy really belong to two separate

[1] Mentally unsound equals insane or idiotic.
[2] Topographie Méd. de la Belgique, Bruxelle, 1865.
[3] Handbuch f. Geograph. und Historische Pathologie.

classes, namely, those which come on directly or indirectly as the result of the disease, and those which arise during its course as they arise in ordinary life.

Naturally enough, a very common variety of complication is some traumatism, severe or mild, and which is suffered as the result of the fall accompanying the fit, whereby the head is struck against some hard or sharp object. Of course the severity of the injury is never the same and cannot be described, for it may be anything from fracture to a slight abrasion or bruise. When such an accident does happen, it pursues the same course as in healthy persons, but it should not be forgotten that the coma of the fit may be dangerously deepened by the concussion, and also that the coma may mislead the physician so that it be regarded not as the natural sequence of an attack, but as produced by the injury. Fractures of the clavicle are very common in these persons, owing to the fact that in falling this bone is suddenly strained by the shoulder striking on the ground or surrounding bodies. In the same manner various dislocations may ensue. The presence of a fracture in an epileptic is a very much more serious matter than would appear at first glance, for even if the fits are not very frequent they are almost sure to cause a fresh solution of continuity, or even to convert a simple into a compound fracture by the jerkings of the muscles. Splints are, of course, of value, and the limb may be wrapped in a pillow, but still disturbance of the part may occur. Careful watching with quiet rest in bed must always be insisted upon, since, under these circumstances, no second fall takes place on the advent of a new attack. A watcher may sometimes be appointed who will use nitrite of amyl whenever a fit seems imminent, but this is hardly practicable in most cases. It is

unnecessary for me to remark that all the secondary changes, produced by injuries in the brain and elsewhere, are to be looked for.

In other cases apoplexy may occur, due to the sudden strain upon the cerebral blood-vessels during the fit, and if the coma following an attack is prolonged or peculiar this fact should be called to mind. The inequality of the pupil, the stertorous respiration, the fact that the tongue cannot be protruded straight from the mouth, all point to cerebral trouble; but the rise of temperature, the coma, and, last of all, the hemiplegia, are characteristic of both states, and cannot be used for differential diagnosis.

Meningitis may also arise, particularly in the cases where the fits have been prolonged and frequent, or it may be caused by the injury received in the fall or tossings.

The frequency of paralysis has already been discussed, under the heading of After-Symptoms. It may be mentioned here, however, that Reynolds has only seen it as a continuous condition in 3 out of 81 cases of epilepsy.

The influence of pregnancy on epilepsy is another point of interest and dispute, some claiming that the fits are increased while others say they are diminished in frequency. Tyler Smith[1] has seen only 2 cases of epilepsy occur during labor in 53 deliveries of 15 epileptic women.

The susceptibility of epileptics to infections diseases is very slight, according to the best authorities. Romberg states it to be very much decreased, and many others agree with him. On the other hand, Esquirol believed epileptics to be singularly open to such diseases.

[1] Lancet, xxiv, p. 644, 1849.

The influenee of aeute diseases on epilepsy has been quite reeently studied by Bourneville and Bonnaire[1] during an epidemie of measles in the epileptics and idiots at the Bieêtre, and they find that during the eourse of the intereurrent malady the fits are much deereased in foree and frequeney. Séglas[2] has also made a series of observations at the Salpêtrière and the Bieêtre, and he reaehes the following eonelusions:—

1. Intercurrent diseases have in the greater number of eases a favorable influence on epilepsy.

2. In some eases this is only during the intereurrent disease.

3. Febrile disorders modify it most eommonly.

[1] Le Progrès Méd., 1883.

[2] Compt. rendu Service Bieêtre from l'armée. Paris, 1883.

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